A STUDY ON DUODENAL ULCER PERFORATION : RISK FACTORS AND PROGNOSTIC DETERMINANTS

A Prospective Study

Dissertation submitted to

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY

in partial fulfillment of the regulations for the award of the degree of

M. S. GENERAL SURGERY (BRANCH I)

CHENGALPATTU MEDICAL COLLEGE

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY

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MAY 2018

CERTIFICATE

This is to certify that this dissertation titled **"A STUDY ON DUODENAL ULCER PERFORATION : RISK FACTORS AND PROGNOSTIC DETERMINANTS IN CHENGALPATTU MEDICAL COLLEGE "** has been prepared by **DR. VAITHISWARAN.A**, under my supervision in the Department of General Surgery, Chengalpattu Medical College, Chengalpattu, during the academic period 2015 – 2018, and is being submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the University regulation for the award of the Degree "Master Of Surgery" (M. S., General Surgery) and his dissertation is a bonafide work.

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DECLARATION

I, **Dr. VAITHISWARAN.A** , solemnly declare that the dissertation "**A STUDY ON DUODENAL ULCER PERFORATION : RISK FACTORS AND PROGNOSTIC DETERMINANTS IN CHENGALPATTU MEDICAL COLLEGE**" is a bonafide work done by me in the Department of General Surgery, Chengalpattu Medical College, Chengalpattu, Under the able guidance of **PROF. Dr.T.RAGUPATHY., M.S**, Professor , Department of General Surgery , Chengalpattu Medical College , Chengalpattu .

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ABBREVIATION

SC with OP	:	Simple Closure with Omental Patch.
Live OPC	:	Live Omental Patch Closure.
TVGJ	:	Truncal Vagotomy with Gastro Jejunostomy.
POD	:	Post Operative Day.
GA	:	General Anaesthesia.
LA	:	Local Anaesthesia.
DM	:	Diabetes Mellitus.
IHD	:	Ischemic Heart Disease.
HT	:	Hypertension.

INSTITUTIONAL ETHICAL COMMITTEE

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Designation	:	Post graduate in General Surgery,
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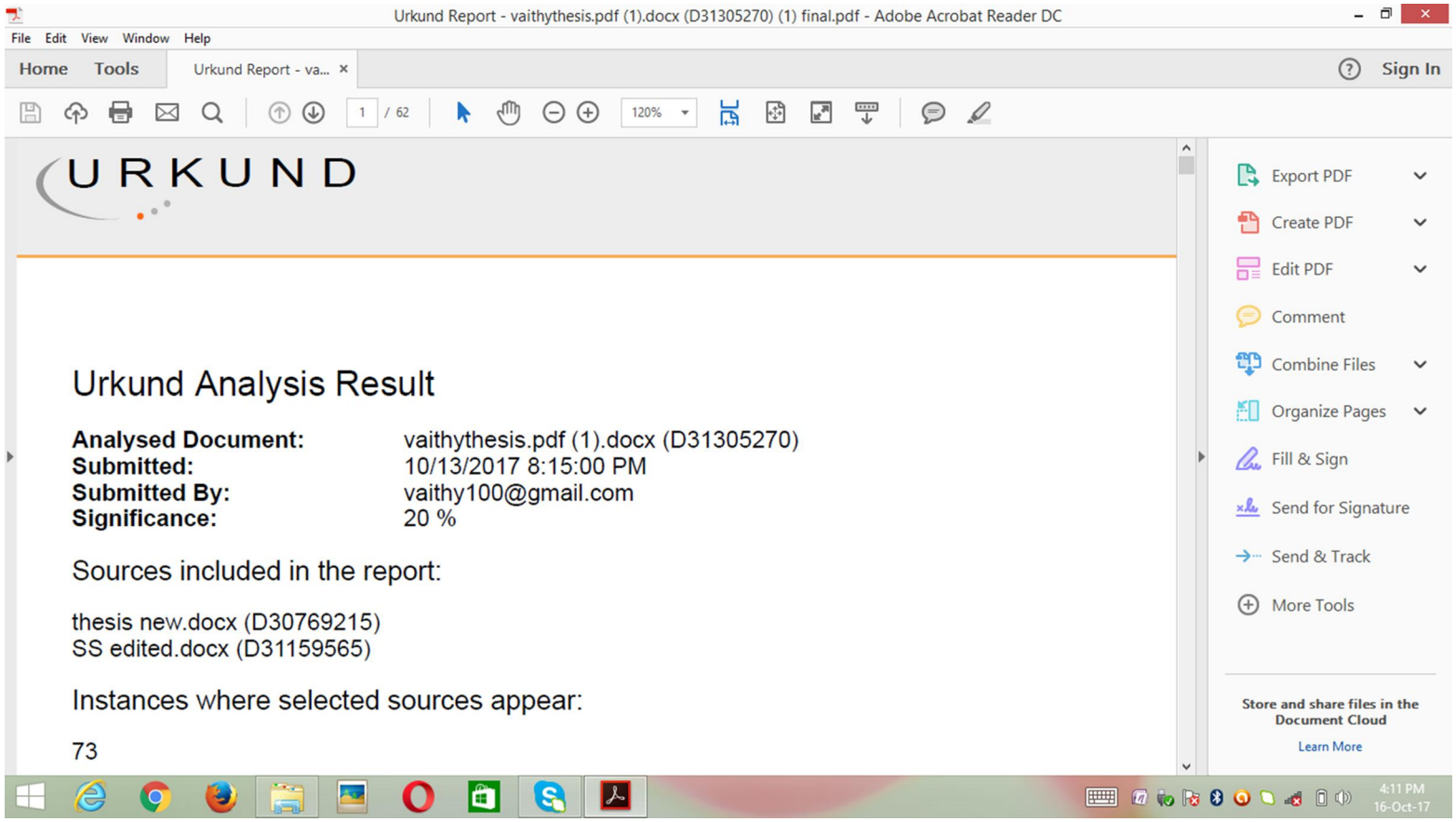
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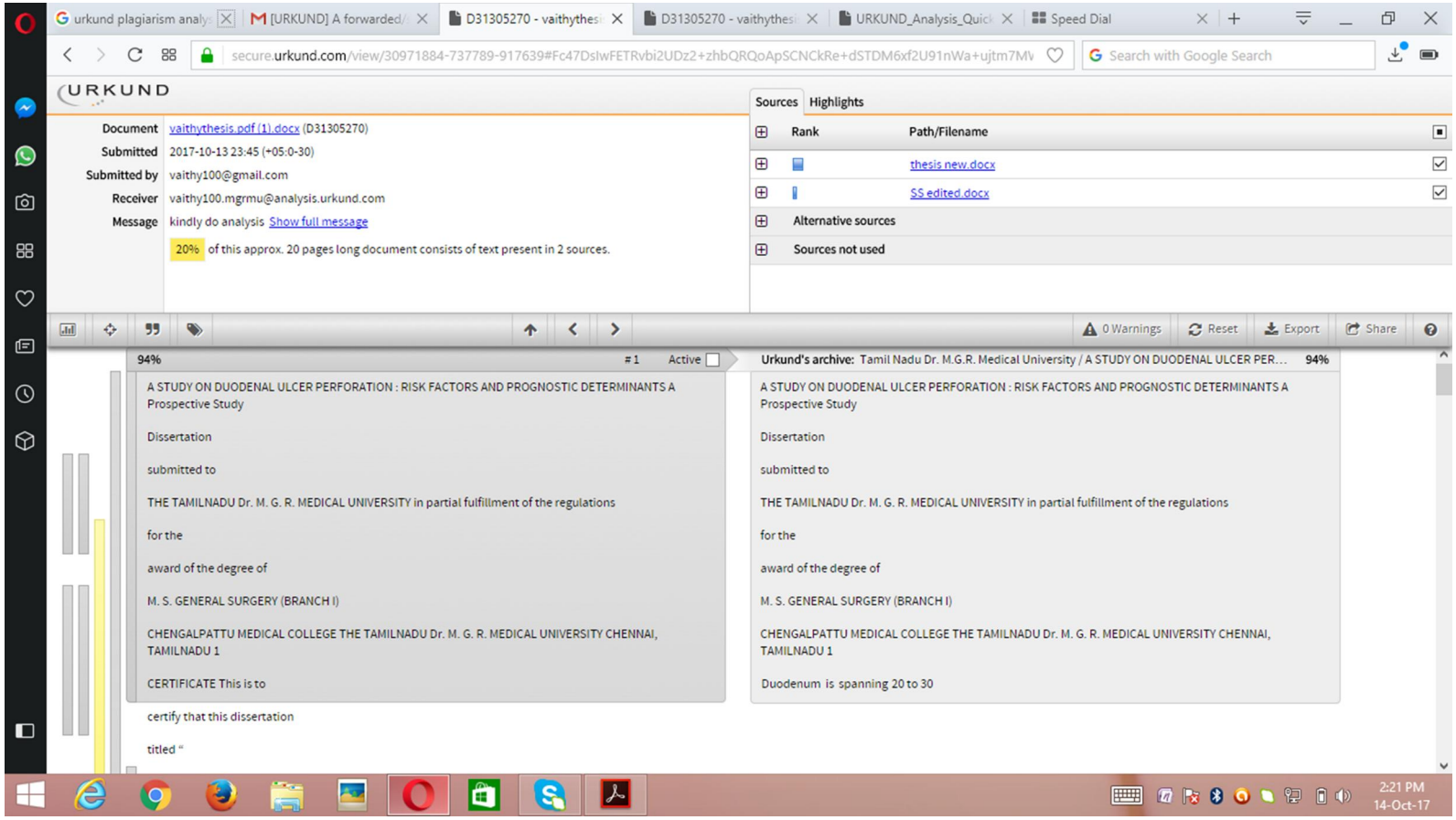
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Duodenum is spanning 20 to 30
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CERTIFICATE II

This is to certify that this dissertation work titled **"A STUDY ON DUODENAL ULCER PERFORATION : RISK FACTORS AND PROGNOSTIC DETERMINANTS**'' of the candidate **DR. A. VAITHISWARAN** with registration Number **221511254** for the award of **degree of M.S.** in the branch of **GENERAL SURGERY (BRANCH – I).** I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **20%** percentage of plagiarism in the dissertation.

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INTRODUCTION

Duodenum is spanning 20 to 30 cm from the pylorus to the ligament of Treitz, the duodenum is the gate controlling the passage of food from the stomach to the jejunum. This organ's name was derived from the Latin phrase intestinum duodenum digitorum, or "intestine of twelve digits."

This Latin phrase may have derived from the writings of the Greek physician Herophilus (334 to 280 b.c.) who described the "beginning of the intestines prior to the beginning of the loops" as the dodekadactilon (dodeka "twelve"; dactilon "fingers"). Although it is the shortest segment of small bowel, the duodenum is the initial site of contact between gastric secretions, bile, and digestive enzymes from the common bile duct and the pancreas.

Thus, it plays an important role in the regulation of digestion, absorption of essential micro- and macro-nutrients, and bowel motility. Its relationships to the major structures of the upper abdomen lend it to exposure during a large number of gastrointestinal interventions ranging from gallbladder removal to gastric bypass and colonic resection. As such, it is important to understand both the structure and function of the duodenum as it relates to alimentary surgery.

EMBRYOGENESIS

In the adult, the duodenum's position in the upper abdomen follows the normal development and rotation of the embryonic gut. This process starts at the beginning of the third week of embryonic development with primitive foregut demarcation from the midgut and hindgut. Early in the second month of gestation, the embryologic midgut migrates ventrally to descend into the yolk sac. The apex of this loop is marked by the omphalo-mesenteric duct (yolk sac) with an axis around the superior mesenteric artery, the proximal portion of the yolk sac's primitive blood supply (vitelline artery).

Over the next several weeks, the mid-portion of the intestine elongates faster than the abdominal cavity expands, thereby enlarging the mid-gut loop, which continues to push into the umbilical cord. As this occurs, the mid-gut undergoes a counterclockwise 90-degree rotation around the superior mesenteric artery, forming "prearterial" and "postarterial" halves. Following this rotation, the cranial ("prearterial") segment of mid-gut (future duodenum and proximal small bowel) lies to the right of the caudal ("postarterial") segment (future colon). The cranial ("prearterial") segment continues to elongate into the umbilical cord until the tenth gestational week, after which the mid-gut returns into the abdomen.

The cranial limb migrates back into the abdomen first, causing the duodenum to pass behind the superior mesenteric artery. The caudal limb follows with the cecum and terminal ileum entering last. During its return, the midgut rotates another 180 degrees (total rotation = 270 degrees). At the completion of these movements, the colon is situated anterior to the superior mesenteric artery, and the cecum is located at the level of the iliac crest. From the twelfth week of gestation until after birth, the colon elongates while the cecum remains in its original position.

This colonic growth effectively produces an “ascent” of the hepatic flexure toward the right upper quadrant that seems like a cecal “descent.” Anatomic relations between the duodenum, the liver, and pancreas are also dictated by early development. The future duodenum lies between the transverse septum of the ventral mesentery (the future primordium of the liver, the bile ducts, and the ventral pancreatic bud) and the dorsal mesentery (the future dorsal pancreatic bud).

After mid-gut rotation is complete, the hepatic parenchyma and the intestines proliferate and the dorsal and ventral pancreatic buds fuse, establishing the duodenum’s final anatomic location. Initially, the duodenum is composed of a single layer of endo-dermal cells surrounded by undifferentiated mesenchymal cells. By the end of the fourth week of gestation, the duodenal mucosa begins to proliferate along the ventral wall near the origin of the hepatic diverticulum.

During mid-gut rotation, mesenchymal tissue beyond the first portion of the duodenum increases along the dorsal aspect of the duodenum, fixing it to the retro-peritoneum beyond this point. During fixation, this dorsal mesentery transforms into an avascular plane of loose connective tissue known as the fascia of Treitz (not to be confused with the ligament of Treitz). This plane is entered when lifting the duodenum medially during a Kocher maneuver.

At the end of the third gestational week, the liver primordium, gallbladder, and biliary duct (both originating from the gallbladder bud) arise as a ventral outgrowth from the distal end of the foregut. Later (fifth gestational week), the connecting elements between the hepatic diverticulum and the duodenum form the bile duct and, ultimately, the cystic duct and the gallbladder. Around the ninth week of gestation, rapid hepatic growth occurs secondary to the hematopoietic function of the liver and the formation of multiple hepatic sinusoids. This hepatic growth, combined with the midgut's elongation, push the duodenum below the liver.

At this time, the ventral mesentery produces the lesser omentum, the falciform ligament, and the hepato-duodenal ligament; these structures envelope the portal triad as it extends from the liver. Other structures within the portal triad include the hepatic artery and the portal vein.

Spatially, the portal vein is intricately related to the duodenum during the latter's development. The portal vein develops from the primitive paired vitelline veins that arise in the yolk sac and pass up the body stalk to enter the developing heart. Two extra-hepatic cross-connections develop between the aired vessels: The cranial anastomosis lies behind the duodenum and the caudal anastomosis passes in front of the duodenum.

Normally the cranial retro-duodenal anastomosis persists as the portal vein and the caudal anastomosis disappears. This preduodenal caudal anastomosis can persist as the portal vein, leading to the rare congenital anomaly known as a preduodenal portal vein. At the end of the fourth gestational week, the developing duodenum is joined by a dorsal pancreatic primordium. One week later the ventral pancreatic primordial bud arises at the base of the hepatic diverticulum.

At the end of the sixth week, these two primordia fuse as the ventral pancreas migrates below and behind the dorsal pancreatic segment; these changes form portions of the adult pancreatic head and uncinate process. After fusion, the principal pancreatic ducts fuse, typically with the ventral duct fusing at the mid-portion of the dorsal pancreatic duct. The combination of the ventral duct and the fused mid- to distal dorsal duct becomes the duct of Wirsung.

This duct connects to the common bile duct, contributing to the ampulla of Vater at the site of the common bile duct's entry into the duodenum. After fusion, the proximal portion of the dorsal pancreatic duct (the duct of Santorini) typically regresses as the duct of Wirsung assumes dominance. As the midgut expands and rotates, the duodenum achieves its final location along the retro-peritoneum with the first and second portion located laterally while the remainder traverses inferiorly to the fused pancreatic primordial. Failure of the pancreatic primordia to fuse results in a condition known as pancreatic divisum. In this situation, the ducts of Wirsung and Santorini drain separately into the duodenum.

Annular pancreas may also develop after anomalous pancreatic fusion; in this developmental anomaly, a thin flat band of normal pancreatic tissue surrounds the second portion of the duodenum and connects to either side of the pancreatic head. The ring that forms around the duodenum can cause duodenal stenosis. Although this anomaly is described in children, it may be entirely asymptomatic until discovered as an incidental finding on necropsy.

GROSS ANATOMY

The duodenum is divided into four segments: the duodenal bulb or cap; the second vertical or descending portion; the third horizontal or transverse portion; and the fourth oblique or ascending portion. The duodenum begins at the end of the

gastric pylorus, in the plane of the first lumbar vertebra. Starting at the second portion, it descends in a C-shaped curve around the pancreatic head. The third part of the duodenum lies inferior to the superior mesenteric artery at the level of the second lumbar vertebra; it is situated in the angle formed by the superior mesenteric artery and the aorta where it crosses the midline to join the fourth duodenal segment and later the jejunum.

The duodenum is related anteriorly to the liver and gallbladder; superiorly to the epiploic foramen; laterally Second portion) and inferiorly (third portion) to the pancreatic head; and posteriorly to the common bile duct, portal vein, inferior vena cava, and gastroduodenal artery. It is separated laterally from the inferior vena cava by a small amount of connective tissue.

FIRST DUODENAL SEGMENT (APPROXIMATELY5 cm LONG)

The first portion of the duodenum passes superiorly from the gastric pylorus to the neck of the gallbladder. The proximal half, the duodenal bulb or cap, is mobile; the distal half is fixed. Most (90%) duodenal ulcers occur in the duodenal bulb. Clinically, the mobility of the duodenal bulb facilitates operations on the pylorus and duodenum, particularly after a Kocher maneuver. Its longitudinal muscle folds can be appreciated on upper endoscopy and used as a landmark prior to entering the second portion where transverse folds can be seen.

The hepatoduodenal portion of the lesser omentum attaches to the superior duodenal border within the initial 2.5 cm of this segment; the greater omentum attaches to this segment's inferior border. The distal 2.5 cm is covered by peritoneum anteriorly, resulting in the posterior surface closely contacting the portal triad and the gastroduodenal artery. This segment's relationship to the gastroduodenal artery explains the artery's susceptibility to bleeding when posterior peptic ulcers erode. When encountering this condition, surgeons should remember that the gastroduodenal artery arises 15 to 30 mm above the superior border of the first part of the duodenum, and the distance between the artery's origin and the pylorus can range from 5 to 50 mm.Finally, the duodenum's proximity to the gallbladder facilitates cholecystoduodenal fistulas and the passage of gallstones into the intestinal tract after severe bouts of cholecystitis.

SECOND DUODENAL SEGMENT (APPROXIMATELY7.5 cm LONG)

This portion of the duodenum extends from the gallbladder neck to the upper border of L4. It joins the first portion of the duodenum on the right side of the first lumbar vertebra, behind the costal margin slightly superior and medial to the ninth costal cartilage's tip. Beyond this junction, the duodenum becomes a retroperitoneal structure through fusion of its lateral visceral peritoneum to the posterolateral abdominal wall. After forming an acute angle with the superior duodenal flexure, the second portion descends from the gallbladder with a loop that

passes over the right renal hilum, the adrenal gland, the psoas major, and the edge of the inferior vena cava.

Concurrently, it passes under the right hepatic lobe, the colonic hepatic flexure, and parts of the transverse colon and the jejunum. Peritoneal folds pass above and below this duodenal segment to form the mesocolon. The relationship between the duodenum, hepatic flexure, and mesocolon must be considered when mobilizing the hepatic flexure during surgical interventions on the proximal colon. Medially, the pancreatic head is intimately related to the duodenal C-loop.

The superior pancreaticoduodenal branch of the gastroduodenal artery runs in the groove between the two structures. At about the midpoint of the C-loop, the pancreaticobiliary tract opens into the papilla of Vater, on the second duodenal segment's concave posteromedial side.

THIRD DUODENAL SEGMENT (APPROXIMATELY 12TO 13 cm LONG)

The third portion of the duodenum extends from the right side of L3 or L4 to the left side of the aorta. As this segment passes from right to left across the midline anterior to the ureter, the psoas muscles, the inferior vena cava and the aorta, it remains posterior to the superior mesenteric vessels. Superiorly, the pancreatic head and uncinate processes are separated from this part of the duodenum by a groove containing the inferior pancreaticoduodenal artery. This

segment ends to the left of the third or fourth lumbar vertebra, next to the root of the small intestine's mesentery.

FOURTH DUODENAL SEGMENT(APPROXIMATELY 2.5 cm LONG)

The fourth portion of the duodenum starts at the left upper border of L2. After an upward and oblique ascent, it travels to the duodenojejunal angle at the root of the transverse mesocolon, approximately 4 cm below and medial to the ninth costal cartilage's tip. It then descends leftward to form the duodenojejunal flexure where the duodenum's suspensory ligament attaches to the mesentery (ligament of Treitz). This ligament, a remnant of the dorsal mesentery, extends from the duodenojejunal flexure to the right diaphragmatic crus. Its termination closely approximates the terminal part of the inferior mesenteric vein, the left ureter, and the left kidney.

ARTERIAL SUPPLY

The first portion of the duodenum is supplied by the posterior superior pancreaticoduodenal branch of the gastroduodenal artery, and variably by the supraduodenal and retroduodenal arteries (either separately or in variable combinations). In some patients, the first centimeter is also supplied by branches of the right gastric artery.

The gastroduodenal artery descends between the first part of the duodenum and the pancreatic head, terminating into the right gastroepiploic artery and the anterior superior pancreaticoduodenal artery.The rich periduodenal rterial anastomotic network often frustrates attempts to control bleeding from posterior duodenal ulcers. The arterial supply to the remainder of the duodenum is derived from major arterial anastomoses between the celiac and superior mesenteric arterial circulations.

As noted earlier, the anterior superior pancreaticoduodenal artery arises from the gastroduodenal artery on the pancreas' ventral surface. The posterior superior pancreaticoduodenal artery crosses in front of the common bile duct and then spirals posteriorly to the pancreatic head. The anterior and posterior inferior pancreaticoduodenal arteries arise from the superior mesenteric artery or its first jejunal branch, either separately or through a common origin.

These two arteries split and run in posterior and anterior grooves between the descending and transverse portions of the duodenum and the pancreatic head where they join to form continuous anterior and posterior arcades. Through these arterial arcades, the duodenum shares its blood supply with the proximal pancreas. As such, resection of either the duodenum or the pancreas alone is technically challenging and potentially hazardous.

VENOUS SUPPLY

Pancreaticoduodenal veins parallel the pancreaticoduodenal arteries, accompanying them in anterior and posterior pancreaticoduodenal arcades.Surgeons usually encounter these veins superficial to their arterial analogues. The lower portion of the proximal duodenal bulb drains into the right gastroepiploic veins; the upper part drains into the portal vein or posterior superior pancreaticoduodenal vein via several suprapyloric veins.

The posterior arcade ends in the portal vein above and the superior mesenteric vein below. The posterior superior pancreaticoduodenal vein may follow its companion artery anterior to the bile duct, although it usually runs behind the duct. This vein terminates inferiorly on the superior mesenteric vein's left border. Here it may be joined by a jejunal vein or by the anterior inferior pancreaticoduodenal vein.

LYMPHATIC DRAINAGE

The lymphatic drainage of the duodenum generally parallels its vasculature. Anterior lymphatic channels drain to anterior pancreatic nodal basins and posterior channels drain to basins posterior to the pancreatic head. Although primary duodenal carcinomas may invade the pancreas via direct extension or lymphatic infiltration, they usually spread to the periduodenal lymph nodes and liver first.

INNERVATION

The duodenum's extrinsic innervation is parasympathetic, arising from the anterior and celiac vagal branches, and sympathetic, arising from the splanch-nic nerves of the celiac ganglion (T6 to T12). Intrinsic innervation arises from the Auerbach myenteric and the Meissner submucosal plexuses. Processes from these neurons innervate their targets (e.g., smooth muscle, secretory and absorptive cells), but also connect to sensory receptors and interdigitate with other neural processes arising from both inside and outside the plexuses.

HISTOLOGY

The duodenal wall is made up of four layers: the serosa: an outer peritoneal coat; the muscularis: a muscular coat made up of longitudinal and circular fibers; the submucosa; and the mucosa that forms its inner lining. The serosa is an extension of the peritoneum. It consists of a single layer of flattened mesothelial cells overlying loose connective tissue. The portions of the posterior and lateral duodenal walls that are retroperitoneal lack this peritoneal or serosal coat.

The muscularis is composed of two layers of smooth muscle: an outer (longitudinal) layer and an inner (circular) layer. The myenteric plexus of Auerbach lies between these two layers. The Meissner plexus is found in the

submucosa along with a network of loose connective tissue rich in lymphatics and small blood vessels.

The glands of Brunner, a characteristic histologic feature of the mammalian duodenum, are found in the sub mucosa; these glands empty into the crypts of Lieberkühn through small secretory ducts.Brunner gland secretion is viscous, alkaline (pH 8.2 to9.3), and clear. These mucoid, viscous, alkaline secretions help protect the duodenal mucosa against the corrosive action of gastric juice.The intestinal mucosa is thrown into numerous finger like projections, or villi, that greatly increase the mucosal surface area. The villi's epithelium is lined by columnar cells containing both mucus andHCO_3secreting surface cells and absorptive cells.

The mucosa lining the crypts and villi can be divided into three layers: the muscularis mucosae (deep); the lamina propria (middle); and an inner layer consisting of a continuous sheet of columnar epithelial cells.The crypt epithelium's main functions include (1) cell renewal; (2) exocrine, endocrine, water, and ion secretion; and (3) absorption of salt, water, and specific nutrients.The crypt epithelium is composed of at least four distinct cell types: Paneth, goblet, undifferentiated, and endocrine cells.

PHYSIOLOGY

The main functions of the duodenum are to

(1) alkalinize acidic chyme, thereby protecting its mucosa and facilitating digestion,

(2) absorb calcium and iron,

(3) further the breakdown of food products, and

(4) exert neuro-endocrine control of upper GI motility and secretion.

ALKALINIZATION AND DUODENALMUCOSAL DEFENSE

Duodenal luminal pH can fluctuate rapidly between pH 2 and 7 as secreted bicarbonate and gastric acid mix. Therefore, prevention of mucosal damage requires a coordinated defense via regulated premucosal, mucosal, and submucosal components. These components include mucus and bicarbonate ($HCO3^-$) secretion, intracellular buffering, neuronal activation, and increased blood flow. After duodenal luminal acidification, a number of compounds stimulate bicarbonate secretion from the liver, pancreas, and duodenum including secretin, vagally produced acetylcholine, vasoactive intestinal polypeptide (VIP), pituitary adenylate cyclase-activating polypeptide (PACAP), melatonin,and motilin.This process may also be mediated by a feedback loop involving luminal adenosine

triphosphate and intestinal alkaline phosphatase activity.The principal components contributing to duodenal mucosal defense, however, involve HCO3 secretion from the epithelium within the duodenal bulb. Duodenal mucosal bicarbonate secretion is stimulated by a complex array of mediators that lead to a net influx of acid from the duodenal lumen into the extracellular space, and a net efflux of extracellular HCO3 into the lumen. The process is thought to start with neutralization of luminal H+ by secreted HCO3. This process is facilitated by extracellular, membrane-bound carbonic anhydrase.A decrease in intracellular pH promotes H+ extrusion into the subepithelial space via basolateral Na+/H+ exchanger 1 (NHE1) activity and movement of extracellular bicarbonate into the cell through basolateral Na+/HCO3 transporter channels (NBC). This new intracellular HCO is then secretedinto the duodenal lumen via brush-border Cl−/HCO3anion exchangers in conjunction with cystic fibrosis transmembrane conductance regulators (CFTR).

Although duodenal mucosal bicarbonate secretion is believed to be one of the primary duodenal mucosal protective mechanisms, additional mechanisms are beginning to emerge, including the receptor potential vanilloid 1 ion channels (TRPV1). Activation causes the release of enteroglucagon, calcitonin gene-relatedpeptide, and nitric oxide, all of which act to simulate mucus secretion and increase blood flow. Concurrently,increased cyclooxygenase activity leads to a delayed increase in mucus production and bicarbonate secretion through a cyclic

adenosine monophosphate- andCa2+-mediated process involving the unique duodenal G-protein-coupled receptors EP3 and EP4.Capsaicinsensitiveafferent nerves and cyclooxygenase activity are also activated by nutrient-specific sensors for l-glutamate,the nutrient conferring “umami” taste and the main free amino acid in dietary protein. l-Glutamate also independently activates gastric vagal afferent nerves leading to the release of nitric oxide and 5-hydroxytryptamine

(5-HT), both of which increase luminal mucous gel thickness and intracellular pH.

Cumulatively, these factors stabilize the pH gradient,increase nitric oxide-mediated vasodilation and subsequent regional blood flow, and increase mucus production by goblet cells and Brunner glands. The latter effect creates a thickened mucous gel lining composed of water,mucin glycoproteins, bicarbonate, and trefoil factor family peptides. This thickened mucous lining creates azone of low turbulence that buffers the duodenal lumen,allowing a small amount of bicarbonate to help neutralize large amount of gastric acid.

In general surgical field, duodenal ulcer perforation is one of the most common acute-abdominal emergency.

But the incidence of duodenal ulcer disease has been declining for past two decades and the need for elective ulcer surgery is also on decline, neither the incidence nor the need for surgery for the emergent complications of ulcer

(perforation, bleeding and obstruction) have changed significantly during the past 15-20 years[1].

The present study deals with one of the complications of peptic ulcer, namely duodenal ulcer perforation, trends in age distribution of occurrence, risk factors, seasonal variation,outcome of operative and non-operative modalities of treatment and factors influencing the prognosis of the disease.

Good history taking, methodical physical examination and good clinical acumen is must in diagnosing the acute abdominal emergency.

Multiple factors has been suggested for influencing the progress of the disease and its prognosis which would be discussed in detail in this study.

AIM OF THE STUDY

The aims of the study are-

1. To study the age group, duodenal ulcer perforation commonly occurs.

2. To study the risk factors and seasonal trends.

3. To study the prognostic factors influencing the disease process.

4. To study the outcome of operative and non—operative treatment, with its morbidity and mortality.

REVIEW OF LITERATURE

Perforated duodenal ulcer is one of the important acute abdominal emergency. Due to perforation of duodenal ulcer, the spillage of gastro-intestinal contents into the peritoneal cavity resulting in peritonitis, fluid and electrolyte imbalance, circulatory insufficiency, septicemia and finally death.

DUODENAL ULCER PERFORATION

The decades of surgical therapy dominating the treatment of PUD have been followed by a period of potent acid-reducing medication use that has now been replaced with a short-term regimen targeting the elimination of *H. pylori* infection. Discussions of the best operation have been replaced with a discussion of the best drug combination to treat the various manifestations of both gastric and duodenal peptic ulceration. A 10-day to 2-week course of drug therapy directed against *H. pylori* has an ulcer recurrence rate equivalent to TV with pyloroplasty. Although emergency operations for both peptic ulcer bleeding and perforation are still occasionally required, even their incidence is on the wane.

Consequently, an entire generation of surgeons is being trained with little experience in the occasional operation needed to treat this disease and have little experience in dealing with the long-term complications that are seen in patients who have undergone operative therapy in the past. Although the introduction of

histamine H2-receptor antagonists in 1977 radically changed the need for elective surgical therapy of PUD, it was the discovery of the association of *Campylobacter pyloridis* (renamed *Helicobacter pylori* in 1989) with peptic ulceration by Warren an Marshall in 1982 that truly revolutionized our understanding of ulcer pathogenesis and its treatment. They received the Nobel Prize for this work in 2005. Epidemiology studies revealed a strong association between *H. pylori* infection and both gastric and duodenal ulcer disease.

Treatment of the infection resulted in long-term cure of peptic ulcers. Despite the development of potent antiacid secretory drugs and treatment for *H. pylori* infection, PUD remains an important clinical problem because of the widespread use of NSAIDs. The cause of peptic ulcers is complex and multifactorial, as they result from the interplay of the effects of gastric acid and pepsin and the gastric mucosal barrier. Any entity that either increases acid and pepsin secretion or weakens the mucosal barrier can result in ulcers.

INCIDENCE:

The incidence of peptic ulcer perforation is approximately7 to 10 cases per 10 lakhs population per annum. Perforation is present in about 7% of patients hospitalized for peptic ulcer disease, and it is the first manifestation of the disease in about 2%of patients with duodenal ulcer disease. After the diagnosis of duodenal ulcer, 0.3% of patients perforate annually in the first 10 years.

Eradication of H.Pylori will reduce this incidence is as yet unknown due to poor study backup. In the duodenum , the ulcers that perforate are usually located anteriorly, and the aphorism that “anterior ulcer usually perforates, posterior one often bleeds” is as relevant today as ever.

EPIDEMIOLOGY:

There was a considerable change in the epidemiology of occurance of perforated peptic ulcer over the past 2 decades.

AGE:

Increasing use of NSAID have resulted in an increased incidence of peptic ulcer perforation in 6th and 7th decades of the life[3].Previously peptic ulcer perforation more common between the age group of thirty to fifty years.

SEX:

Previously peptic ulcer perforation male to female ratio is 2:1 but nowadays perforation of duodenal ulcer are more common in elderly female patients.

OCCUPATION:

Peptic ulcer perforation more common in lower socio-economic status.

SEASON:

The Peptic ulcer perforation is more common in winter season(December, January and February).

RISK FACTORS FOR DUODENAL ULCER PERFORATION:

A strong association was observed between the use of non-steroidal anti-inflammatory agents (NSAIDs) and perforation of duodenal ulcers. The use of NSAIDs appears to be the major precipitating factor in currently treated patients[3]. A second risk factor for perforation is immuno-suppression, particularly among organ transplant patients treated with steroids. Other factors include increasing patient age, multiple organ system failure ,chronic obstructive lung disease, and major burns.

PATHOPHYSIOLOGY

Peptic ulceration results when the effects of pepsin and acid in the gastrointestinal lumen over-whelm the ability of the mucosa to resist and protect from those effects.

The gastro-duodenal mucosa is exposed to corressive effect of acid and pepsin continuously, but ulceration is an abnormal event. The mechanisms by which it normally enable the mucosa to resist acid-peptic attack can be divided into

three important components-pre-epithelial, epithelial, and post-epithelial defense mechanisms.

DEFENSE MECHANISMS- PRE-EPITHELIAL

Gastric and duodenal epithelial cells are normally are protected from acid-peptic attack by a prominent coat of mucus and by a layer of unstirred fluid that is rich inbicarbonate[4]. Bicarbonate from the blood also enters the unstirred water layer through the process of para-cellular diffusion. Both mucus and bicarbonate are secreted into the lumen by gastric epithelial cells and by Brunner's glands in the duodenum .

Glycoproteins within the mucus layer form a physical barrier to the diffusion of pepsin, and the bicarbonate ions that accompany the glycol-proteins can neutralize acid. Secreted mucus also contains surface-active phospholipids that are secreted by epithelial cells and these phospholipids may protect the mucosa by forming a hydrophobic layer that repels acid at the luminal surface of the mucus gel. The pH on the surface of the gastro-duodenal epithelial cell normally maintained in the neutral range, even when pH in the lumen falls below 2.8.

Acid- peptic injury to the gastro-duodenal mucosa result in an outpouring of mucus, fibrin, and cellular debris that form a protective cap which prevents injured epithelium and impedes further contact with acid. The pre-epithelial defense

mechanisms abnormality may contribute to peptic ulcer disease.

H. pylori infection can be associated with abnormalities in gastro-intestinal mucus and in gastro-duodenal bicarbonate secretion that predispose to peptic ulcer disease[5].

DEFENSE MECHANISMS -EPITHELIAL

Epithelial mechanisms can prevent or minimize acid-peptic injury caused by pepsin and acid breach in the pre-epithelial defense. The surface cells has apical cell membranes and the tight junctional complexes between which act as a barrier that limit the diffusion of hydrogen ions into the mucosa.

The apical membranes exposed to dilute acid causes an increase in its resistance to the passage of hydrogen ions through the tight junctions, whereas exposure to more concentrated acid (pH < 2.5) induces injury that allows hydrogen ions to leak through this para-cellularpathway[6]. The ion pumps in the epithelial cells prevents entry of hydrogen ion into the baso-lateral cell membrane that include a Na+/H+ exchanger and a CI-/HCO-3exchanger.

Duodenal epithelial cells, which also have a Na/HCO-3co-transporterthat helps to regulate intra-cellular pH. Injury to superficial mucosal defects can be sealed quickly through a process called rapid restitution in which healthy cells in the mucous neck region of the gland migrate along the basement-membrane to

close the mucosal gap, which is regulated by growth factors such as epidermal growth factor and fibroblast growth factor. Regeneration by cell-division is also regulated by growth factors.

DEFENSE MECHANISMS- POST-EPITHELIAL

Blood flow which comprise it, provides much of the energy and the substrates necessary both for maintaining epithelial cell integrity and for protective epithelial cells. Blood flow removes the acid that diffuses through an injured mucosal cells into the epithelial cells. The Blood flow transports the $HC0_3$, the alkaline tide to the surface epithelial cells.

The Peptic ulcer disease occurs when the caustic effects of acid and pepsin in the gastro-intestinal lumen as abnormality in the defense mechanism.

PEPTIC ULCER DUE TO NSAID:

Majority of peptic ulcerations is due to NSAID intake. Peptic ulceration with NSAIDs typically asymptomatic, but NSAID-induced duodenal ulcers can be symptomatic further complicated by GIT bleeding, perforation, and obstruction. Peptic ulcerations can be documented endo-scopically in 15% to 45% of patients on chronic NSAID therapy who are asymptomatic. Though one to four percent of patients receiving NSAIDs for 1year will experience serious GIT complications[7].

PATHOPHYSIOLOGY OF NSAID ULCERS

The patho-physiology divided into two categories: those are cyclo-oxygenase inhibition dependent and those independent of cyclo-oxygenase inhibition. The cyclo-oxigenase independent is due to local mucosal toxic processes, which by attenuation of the phospholipid content and surface hydro-phobicity of the gastric mucus layer. Local topical effects of NSAIDs are responsible for the acute hemorrhages and erosions. Following NSAID ingestion surface epithelial cells are damaged and increased mucosal permeability occur. Almost NSAIDs are weak acids that in acidic gastric juice are un-ionized and become lipid soluble. These lipid-soluble NSAIDs diffuse across gastric mucosal epithelial cell where they ionize at neutral pH and then trapped within the cells which is responsible for local toxic effects. Advanced age is also a substantial risk factor.

Some studies have found that the risk of NSAID-associated gastro-intestinal complications is more during the first 30 days of NSAID use.The dose of an NSAID increases, the risk of ulcers complications also increases. Other risk factors are con-comintant use of gluco-corticoidsor anti-coagulants and co-morbid conditions .

The H. pylori infection and NSAID use may be a additive risk factors for peptic ulcer disease by two-fold increased risk for developing bleeding peptic ulcers compared with uninfected NSAID users. The low-dose aspirin also increases gastric musosal injury in H. pylori infected persons than uninfected ones.

ACID HYPERSECRETORY STATES AND ULCER DISEASE:

Both Zollinger-Ellison syndrome, as a consequence of gastrinoma, and retained gastric antrum after antrectomy with gastrojejunal anastomosis (so-called retained excluded antrum) result in peptic ulceration secondary to high levels of gastrin secreted by the G cells of the gastric antrum. In cases of retained excluded gastric antrum, the residual gastric antral tissue is constantly bathed in a fluid with a high pH (nonacid), resulting in continuous secretion of gastrin. Fortunately, because of the infrequency of antrectomy in today's surgical practice, this clinical situation is rarely encountered. In both disease states, high levels of serum gastrin result in gastric acid hypersecretion and resultant peptic ulceration. Serum gastrin elevations are also seen in chronic atrophic gastritis as a consequence of the lack of gastric acid secretion (typically achlorhydria) causing chronic G-cell stimulation. In Zollinger-Ellison, patients may present with virulent or atypical ulcer disease manifested by ulcers in the distal duodenum and often associated with diarrhea.

The diagnosis is made by sampling serum gastrin levels. A serum gastrin level of 1000 pg/ mL is virtually diagnostic of Zollinger-Ellison, while more

moderate elevations may require further study, including a secretin stimulation test. Of note, 68% of patients with Zollinger-Ellison have fasting serum gastrin concentrations between 100 pg/mL and 1000 pg/mL. In these patients, an increase in the serum gastrin level, after administration of secretin, of 120 pg/mL has a 93% sensitivity, and of 200 pg/mL an 85% sensitivity, in diagnosing gastrinoma.

ALCOHOL AS A RISK FACTOR:

The prevalence of ulcer disease is more in alcoholic cirrhosis[10] appears than with drinkers without cirrhosis. In some retro-spective study limited alcohol ingestion can even protect against peptic ulcer disease[11].

DIET:

As per no study provide a link between dietand peptic ulceration but intake of Coffee, tea, and colas are potent gastric acid secreta-gogues but without any study proof. The both caffeinated and de-caffeinated coffee have same potential for stimulation of gastric acidproduction[12].

CIGARETTE SMOKING*:*

Cigarette smoking is the most important potential risk factor for peptic ulcer disease[9]. It also prevents healing process of peptic ulcerations .Cigarette smokers have both descrease in mucus prostaglandin and bicarbonate producing capablity.

PEPTIC ULCER AND CHRONIC ILLNESSES:

Most of the chronic illnesses like cirrhosis[14], CKD, COPD, Cushing's disease, hyperparathyroidism , and coronary artery disease have been associated with peptic ulcer disease. In one study peptic ulcerations have been found in up to thirty percent of patients with chronic pulmonarydisease[13]. The mechanisms not clear, although cigarette smoking may be the reason.

EMOTIONAL STRESS:

The association between emotional stress and Peptic ulcer disease was been the study of interest by many surgeons. Some newer studies suggest that emotional stress contributes to peptic ulcer disease.

GENETICS FACTORS:

Many genetic factors predispose a person to the development of peptic ulcer disease, still specific gene responsible not yet found. Genetically many are susceptible for H.pylori infection may be the reason.The elevated level of serum pepsinogen-I, has concluded initially as a genetic marker for peptic ulcer disease, also appears to be a reversible consequence of H. pylori infection[16]. Few more suggested genetic markers for peptic ulcer disease include O blood group antigen.

The Lewis blood group antigens was reported to mediate H.pylori attachment to gastric mucosa[17].

DUODENAL ULCER PERFORATION- PATHOGENESIS :

With inadequate blood supply there by slough out of ulcer floor occurs lead to peptic ulcer perforation, followed by leakage of gastric or duodenal contents into the peritoneal cavity, responsible for acute diffuse peritonitis. Peritonitis initially is chemical one, with concurrent bacterial contamination which can aggravate the inflammatory process and progress infective peritonitis , and development of intra-abdominal abscesses over days.

The most common and serious complications of acute peritonitis is adynamic intestinal obstruction and bacterial endo-toxins which leads to septicaemia. Followed by endo-toxic shock and fluid with electrolyte imbalance .

BACTERIOLOGY

Organism commonly isolated from peritoneal fluid areE.coli, Streptococci, Staphylococci and Anaerobic Organism[18].

HELICOBACTER PYLORI

In recurrent pepticulcer perforation, H.pylori infections are more common, which is micro-aerophilic, Curved, Gram negative, urease producing bacteria

which used to colonize the gastric mucosa(deep into the overlying mucus layer). Helicobacter pylori exerts its pathological effects by the expression of various enzymes and its toxins locally.The Helicobacter pylori infection is present usually in all patients with duodenal ulcer following metaplasia and about seventy percent of the patients with gastric mucosal ulcer.

More than 50% of people worldwide harbor H. pylori infection but less than 10% of those infected develop PUD. A number of factors determine whether H. pylori infection causes disease: the pattern of histologic gastritis induced; changes in homeostasis of gastrin and acid secretion; gastric metaplasia in the duodenum; interaction of H. pylori with the mucosal barrier; and the strain of H. pylori present. H. pylori colonizes the entire gastric epithelium, from the cardia to the antrum. However, the severity of the chronic mucosal inflammation is variable and the resultant clinical scenario is dependent on the distribution of the inflammation. In patients with duodenal ulcer, density of infection and severity of inflammation are greatest in the distal antral region with sparing of the acid-secreting body mucosa. After H. pylori eradication, the gastric mucosal changes revert to normal. In gastric ulcer, the body and antrum are affected to a similar degree. In this case, gastric acid secretion can be decreased because of the more severe involvement of the parietal cell region. In response to the same stimulation with gastrin, duodenal ulcer patients with H. pylori produce more acid than infected patients without

ulcers. This may result from an impaired acidsecreting ability of the nonulcer H. pylori-infected patient's more diseased acid-secreting fundus mucosa. Increased gastric acid can lead to the development of gastric metaplasia in the duodenal bulb. This is a necessary forerunner to colonization of the duodenal epithelium with H. pylori. The metaplastic, H. pylori-colonized, duodenal epithelium then becomes more susceptible to acid and pepsin effects and ulceration. After the eradication of H. pylori infection, gastric metaplasia in the duodenum does not revert to normal, but with the elimination of the infection, the risk of ulcer recurrence is eliminated.

H. pylori infection impairs the negative feedback of gastrin release by somatostatin secreted by antral D cells. Somatostatin causes inhibition of gastrin release through a paracrine effect. Production of alkaline ammonia by the bacteria on both the surface epithelium and in the antral glands prevents the D cells from properly interpreting the level of acid present. This leads to improperly low levels of somatostatin, and thus loss of gastrin inhibition. Chronic hypergastrinemia caused by H. pylori exerts a trophic effect and hyperplasia of the acid-secreting parietal cells. Infection with H. pylori also interferes with the neural connections between the antrum and fundus that downregulate acid production. This impaired neural control, coupled with hypergastrinemia, leads to further increases in acid

production. With H. pylori eradication, the hypergastrinemia rapidly resolves. Resolution of acid hypersecretion occurs much more slowly.

The inflammatory response caused by H. pylori infection of the gastric mucosa leads to the cytokine production. This, coupled with the release of lysosomal enzymes, leukotrienes, and reactive oxygen molecules from neutrophils and macrophages further damages mucosal defense mechanisms and enhances ulceration. There is a great deal of variation in the virulence of different strains of H. pylori. More virulent strains have increased production of toxic enzymes and tend to be more adherent to the mucosa. They appear to produce more urease that catalyzes the production of ammonia, further enhancing their potential for harm Some genotypes of H. pylori appear to be particularly toxic and are more common in patients with peptic ulcers. These are vacA-positive and cagA-positive. There is also a genetic predisposition to acquire H. pylori infection. This has been demonstrated in monozygotic and dizygotic twins with an increased risk in the monozygotic twins.

Complex interactions occur between H. pylori and host defense mechanisms that affect the occurrence of peptic ulceration. Duodenal ulcers appear to be predominantly related to increased acid production, whereas in gastric ulceration, defense mechanism breaches appear to prevail. Despite these differences in mechanisms, H. pylori eradication effectively cures PUD and prevents relapses. In

addition, the rate of ulcer healing is accelerated if antibiotics effective against H. pylori are given in addition to drugs that suppress acid. This effect is long lasting.

The mucosal barrier is disturbed by the production of an endo-peptidase having a powerful mucolytic action. Antibiotic treatment for Helicobacter pylori infection results healing of peptic ulcers and it tends to prevents their recurrence rate in good percentage. The production of enormous quantity of ammonia by Helicobacter Pylori leads to an alteration of the epithelial surface pH and alteration in cellular permeability, mucosal charge gradient and epithelial Na-K ATPase activity leading to back diffusion of Hydrogen Ions.

Clinically Chromo-Endoscopy and Urease breath test maybe helpful in diagnosis of Helicobacter pylori infection. OGD-scopy biopsy and culture in appropriate media is the gold standard technique. The microscopically organism can be detected using Giemsa stain and Warthin-Starry Silver stain.

CLINICAL FEATURES OF DUODENAL PERFORATION

Perforations are usually classified into

- Acute perforation- Massive
- Sub-acute perforation
- Slow perforation

- Chronic (or) confined perforation.

ACUTE PERFORATION- MASSIVE

Duodenal ulcer , its clinical course can be divided into three stages.

- Primary or stage of chemical peritonitis.
- Secondary or stage of peritoneal reaction.
- Tertiary or stage of bacterial peritonitis.

PRIMARY STAGE

This occurs within the first two to three hours following duodenal ulcer perforation. The patient is pale anxious and difficult to move. Characterised by pain, peripheral vasoconstriction, sweating and inhibition of respiratory movements. May be febrile but the pulse is raised.

On examination abdomen is held still or board like , with restricted mobility or not at all with respiration. Whole abdomen is tender with board like rigidity. It is dull to percussion with rebound tenderness positive. Usually pelvic tenderness can be elicited on digital rectal examination in appropriate position.

SECONDARY STAGE

This stage usually last from three to six hours. Depending on patients

immunity or defence mechanism , sealing of perforation can occur or there will be diffuse chemical peritonitis,with apparent improvements in the general conditions. The Pain, tenderness, rigidity may lessen. Temperature rises and pulse raises higher, the bowel sounds may be absent. There is temporary improvement known as ‘period of illusion’.

TERTIARY STAGE

After six hours the stage of diffuse peritonitis develops accompanied by silent abdominal distension. More free fluid may have collected which may be clinically detected using various clinical demonstrations. The rising pulse rate with reduction in mean arteriolar pressure marks its progressive deterioration in the patient’s general body condition.

SUB-ACUTE PERFORATION:

Duodenal ulcer some times perforate and which may be sealed rapidly (which is a physiological protective process), before contamination occurs in general peritoneal cavity.

Guarding, rigidity wit rebound tenderness in Epi-gastrium and Right hypo-chondrium . Abdomen may be soft. This type of presentation is known as sub-acute duodenal perforation.

SLOW PERFORATION

Pain may be less severe, less generalised with definitive tenderness but with equivocal presentation , that is equivocal guarding and rigidity, where the duodenal ulcer perforation is frequently persistent.

A small amount of fluid may track down into the right para-colic gutter, producing pain in the right iliac fossa region and tenderness in right iliac fossa region, simulating acute appendicitis.

CHRONIC PERFORATION:

When there is full thickness hole is created by an peptic ulcer disease but the spillage is prevented by Contiguous sealing effect of organ such as Colon, greater omentum creating a walled of area. Signs and symptoms are absent or subtle.

PERFORATION AND HAEMORRHAGE

This type of rare presentation of duodenal perforation with hemorrhage that is combination of Haemorrhage and perforation occurs in 3 ways.

- Simultaneous haemorrhage and perforation.
- Duodenal ulcer perforation occurs during medical treatment of haemorrhage.
- Haemorrhage following a recently sutured or operated perforation.

INVESTIGATION

The proper clinical laboratory evaluation of patient ssuspected duodenal ulcer perforation includes a Hemogram or complete blood count, renal function test, serum electrolytes, liver function test and serum amylase. Patients who presents late following duodenal ulcer perforation may require detailed assessment of renal function with a serum creatinine and pulmonary function and acid-base balance with an arterial blood gas analysis for proper intervention.

The leukocytosis with a left shift is usually the presentation in acute inflamation, but may be absent in the immune-suppressed individuals or elderly patients. The Serum amylase will be within normal limits mostly, but elevated levels of serum amylase less than three times than the normal are occasionally encountered. The liver function tests are usually within normal limits as they present to emergency department early, the scenario is different in late presentation as there will be elevated liver parameters. One of the signs of multi organ dysfunction syndrome. Un-less the presentation is delayed, the serum electrolytes and renal function are normal or-else deranged.

X-Ray

A chest x-ray, abdominal erect x-ray in stable patients and supine and left decu-bitusabdominal films in unstable patients should be obtained. Free air or

pneumo-peritoneum in the peritoneal cavity is seen in seventy percent of patients. The absence of free air in the peritoneal cavity, therefore does not exclude the diagnosis of hollow viscus perforation.

When hollow viscus perforation issuspected, but pneumo-peritoneum is absent in x-ray, usally CT-Abdomen or an upper gastro-intestinal study with water soluble contrast may establish the diagnosis of hollow viscus perforation. With clear-cut signs of peritonitis, however such contrast studies are usually unnecessary.

CAUSES OF PSEUDOPNEUMO PERITONEUM:

- Chiliaditi syndrome.
- Sub-phrenic abscess(air producing organism).
- Sub-pulmonary pneumo-thorax.
- Sub-diaphragmatic fat.
- Curvilinear Pulmonary collapse or atelectasis.
- Omental fat inter-position between liver and diagphram.
- Intramural gas in pneumatosis intestinalis.

CONTRAST RADIOGRAPHIC STUDIES:

In some doubtful cases, Gastro-graffin is used to differentiate sealed from un-sealed perforation duodenal perforation but not used nowadays.

USG / CT SCAN ABDOMEN:

Easily detects free intra-peritoneal air and free fluid in peritoneal cavity.

DIFFERENTIAL DIAGNOSIS:

The differential diagnosis for duodenal perforation can be divided intra-abdominal, Intra-thoracic and Metabolic conditions.

Intra- abdominal cause:

- Acute Appendicitis.
- Acute Intestinal Obstruction with Strangulation.
- Mesentric vein or artery thrombosis.
- Acute pancreatitis.
- Acute Cholecystitis.
- Ruptured Ectopic pregnancy.
- Traumatic perforation of hollow viscus.

Intra-thoracic causes

- Pneumonia.
- Pleuritis.
- Spontaneous pneumo-thorax
- Myocardial infarction.
- Emetic rupture of Esophagus.

Metabolic/Neurologic causes

- Acute intermittent porphyria
- Multiple Sclerosis
- Diabetes mellitus.
- Uremia.
- Neuro-syphilis

MANAGEMENT

As per protocol initial management of duodenal perforation starts from the insertion of a Ryles tube, intravenous fluids, and monitoring of urine output viaa bladder catheterization. Intravenous antibiotics, usually a broad-spectrum cephalosporin or for more seriously ill patients, combination therapy with ampicillin, gentamicin, and metronidazole are instituted. Central hemodynamic monitoringis indicated in unstable patients or those with significant cardio pulmonary instability. Prolonged efforts to establish a diagnosis and resuscitate these patients are counter-productive, as early operation is warranted. The main goal of initial management should be to promptly re-establish intravascular fluid volume in order to decrease the risk of the anesthesia induction and recovery.

SURGICAL TREATMENT:

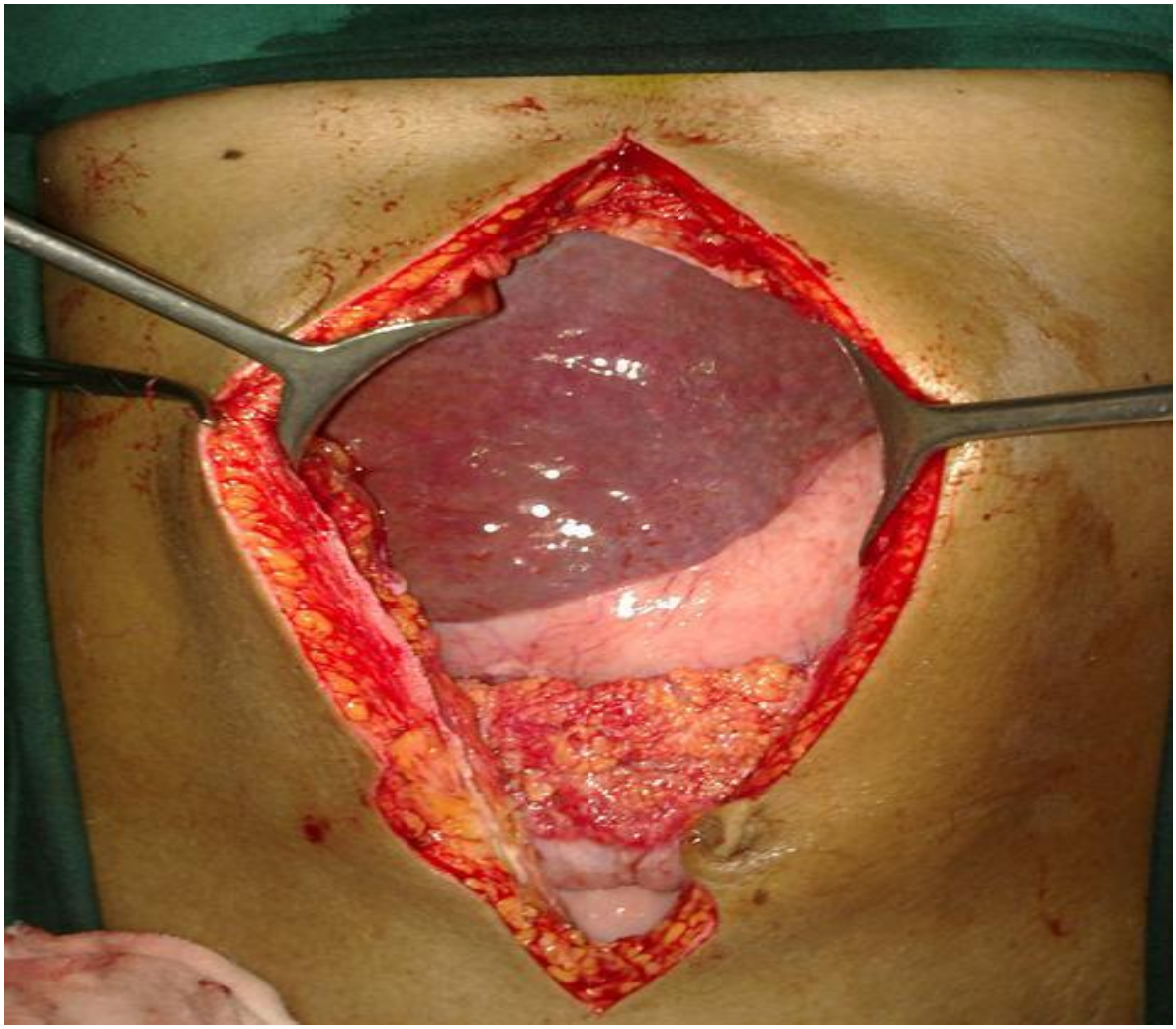
Ideally, the patient with a perforated peptic ulcer should be operated within the hour of initial presentation. Delay in early management of duodenal ulcer perforation ,especially more than twenty four hours, increases mortality and morbidity rates ,of the patient and length of hospital stasis also more.

Various technique available are

- Simple closure of perforation with live omental patch closure.

PICTURE 1: Upper Midline Laprotomy Incision Done.

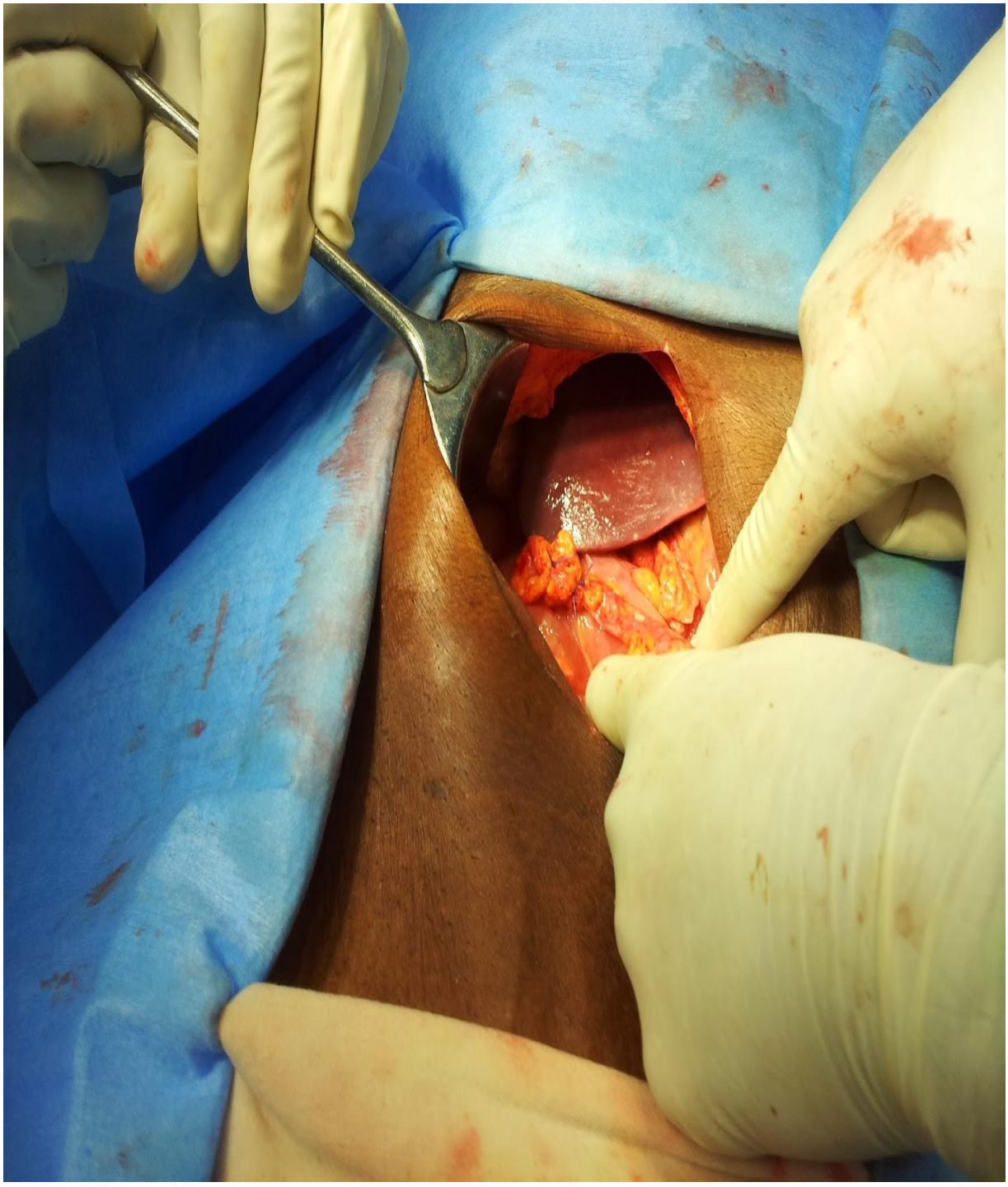
PICTURE 2: Upper Midline Laprotomy Incision With Rectus Opened.



PICTURE 3: Upper Midline Laprotomy Done With Exposing The Visceral Organs.

PICTURE 4: Duodenal Perforation.

PICTURE 5:Duodenal Perforation.



PICTURE 6:Duodenal Perforation Closure-Live omental patch closure .

- Serosal onlay patch closure of perforation.
- Simple closure of perforation with definitive procedure for ulcer.
- Perforated duodenal ulcer closure using Endo-scopy .
- Perforated ulcer using Lapro-scopy.
- Flank drain and
- conservative management.

In our hospital, most commonly done procedure in emergency situation is simple closure with liveomental patch or simple live omental patch closure(modified) technique for the perforated peptic ulcer.

NON-OPERATIVE MANAGEMENT

Occasionally patients present late (> 24 hours after perforation). In this group of patients, non-operative management may be considered if

(1) the patient is hemo-dynamically stable.

(2) generalized peritonitis is absent. and

(3) water soluble contrast examination reveals no leakage of contrast material into the peritoneal cavity.

These types of patients are managed as follows , Ryles tube insertion and suction, PPI'S,intravenous histamine H2-receptor antagonists, broad-spectrum intravenous antibiotics, and close clinical observation[2].

Surgical management should be immediately considered if clinical deterioration occurs. These patients are mostly susceptible to the development of sub-phrenic or sub-hepatic intra abdominal collections turning into abscess.This complications are usually can be managed with conservative or per-cutaneous catheter drainage depends upon size of the abscess cavity.

Previous retro-spective studies have suggested a more liberal use of non-operative management, even in early perforation, as long as no free leak is identifiedand diffuse peritonitis is absent. A recent randomized, prospective trial conducted in Hong Kong confirms that the non-operative management is safe in selected number of patients[19]. Caution should be exercised in application of this approach to the elderly patient. Only one-third of patients who is more than seventy years of age were successfully managed non-operatively in the Hong Kong study series, compared with nearly eighty percent of those forty to seventy years of age, and hundred percent of those younger than forty years of age. Elderly patients are less likely to tolerate complications related to failure of the non-operative approach, and early surgical intervention is preferable.

DEFINITIVE PROCEDURES FOR EARLY DUODENAL ULCER PERFORATION

- Truncal vagotomy with drainage procedure of choice which is suitable.
- Selective vagotomy with suitable drainage procedure.
- Highly selective vagotomy or parietal cell vagotomy.

INDICATIONS FOR DEFINITE ULCER SURGERY*:*

- Duration of perforation less than 24 hours.
- Hemo-dynamically stable, young patients.
- No obvious co-morbid illness.
- Patients with history of long standing peptic ulcer disease.
- Perforation of an ulcer during anti-secretory treatment.
- Previous ulcer complication.

CONTRA INDICATIONS:

Associated medical illness, delay in presentation of more than24 hours.

POST OPERATIVE MANAGEMENT:

H_2-receptor antagonists, proton pump inhibitors should be given for 4-6 weeks. Antibiotic therapy for H.pylori should begiven[20].

POST OPERATIVE COMPLICATIONS:

- Surgical site infection.
- Electrolyte imbalance.
- Intra-abdominal abscess
- Duodenal fistula.
- Gastric outlet obstruction.
- Respiratory complications like basal atelectasis.
- Deep vein thrombosis and pulmonary embolism .
- Bedsore.

PROGNOSIS OF DUODENAL ULCER PERFORATION:

The prognosis will depends on

- The amount and nature of the free fluid in the peritoneal cavity.
- Larger the perforation poorer the prognosis.
- Elderly age group pooer the prognosis.
- Associated medical illness.
- Longer the time lag worse the prognosis.

MATERIALS AND THE METHODS:

This prospective study was conducted in the Department of General Surgery, Chengalpattu Medical College and Government General Hospital during the period of October 2016 to September 2017.The diagnosis of duodenal ulcer perforation was that established by the admitting surgeon, based on clinical feature and supported by radiological evidence and confirmed at operation. Surgery was defined as urgent (less than 4 hours between admission and surgery), same day (4-24 hours) and delayed at a later time during the same hospital admission. Operative details included the site, size of perforation and nature of operation perforation performed. Mortality was defined as death following surgical procedure. Post operative morbidity of the patient was defined in terms of the duration of hospital stay and associated complications following surgery or non-surgical management.

INCLUSION CRITERIA:

1.Out patient and ward patient came to Department of General Surgery, Chengalpattu Medical College and Hospital , Chengalpattu-01, whose age is more than 18 yrs of age.

EXCLUSION CRITERIA:

1. Cases of accidental duodenal perforation during Laparotomy.

2. Cases of gastric antral perforation.

3. Cases of traumatic duodenal perforation.

PROFORMA

Name(in capital letter) :

Age :

Sex : M/F

I.P. No. :

Occupation :

Socio-economic status :

Rural/Urban :

Date of Admission :

H/o. abdomen pain :

Duration of illness :

H/o. Abdominal distension :

H/o. Acid Peptic Disorder :

H/o. Drug intake :

H/o. alcohol intake. :

Previous H/o. any abdominal Surgery :

Systemic medical illness :

EXAMINATION

Pulse Rate

B.P.

Spo2

PER -ABDOMEN

Guarding

Rigidity

Rebound tenderness

Abdominal distension

Obliteration of liver dullness

Bowel sounds

Digital rectal examination

INVESTIGATIONS:

Blood Hb%

Total leucocyte Count

Differential leucocyte count

Blood grouping/ typing

E.S.R.

Blood Sugar

Blood Urea

Serum Creatinine

Serum Electrolytes

Chest-xray

X-ray abdomen

USG –Abdomen

CT-Abdomen

ECG

MANAGEMENT:

Date of Surgery:

Date of Discharge:

Time delay:

Operative findings: Site and size

Acute/chronic ulcer

Nature of peritoneal fluid

Procedure: Simple closure with omental patch or simple live omental patch closure / with definitve surgery/Non operative treatment.

Post-Operative Follow up(complications any)

RESULTS

One hundred and two cases of Duodenal ulcer perforation were studied.

In all 100 cases underwent Laparotomy and the perforation was found in the anterior aspect of first part of the duodenum .

All cases were advised to continue anti-H.Pylori treatment for 4 weeks post-operatively.

The following observations were made out

AGE DISTIBUTION

Age distribution in our study group is

TABLE 1: AGE DISTRIBUTION

Age	No. of Case	Percentage (%)
< 19 yrs	1	0.98%
20 - 29 yrs	22	21.57%
30 - 39 yrs	19	18.63%
40 - 49 yrs	32	31.37%
50 -59 yrs	16	15.69%
> 60 yrs	12	11.76%

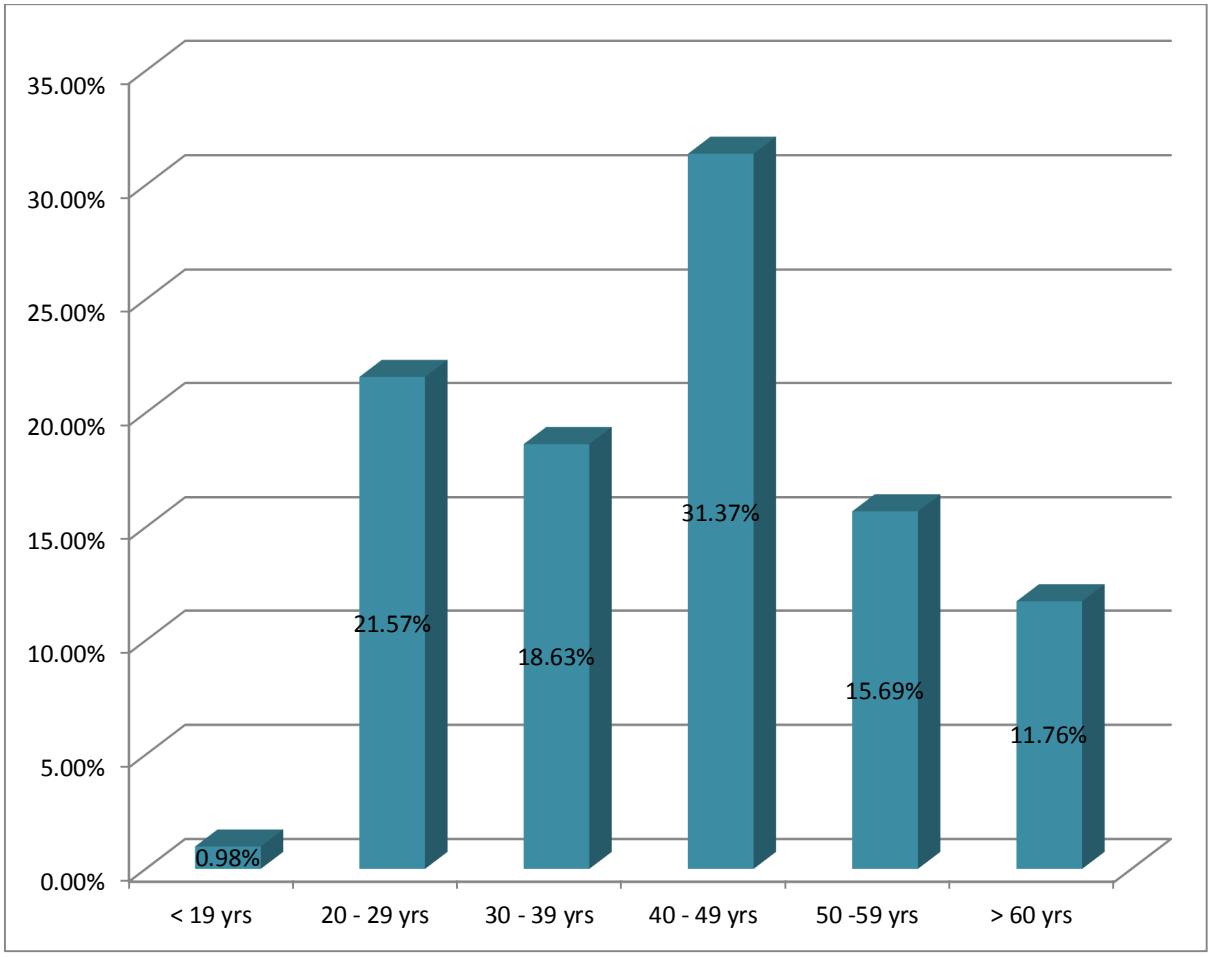


Figure 1: Age Distribution.

SEX

In our study,3 Female patients with duodenal ulcer perforation were noted. Male to Female ratio 33:1.

TABLE 2: SEX DISTRIBUTION.

	No. of Case	Ratio
Male	99	97.06%
Female	3	2.94%

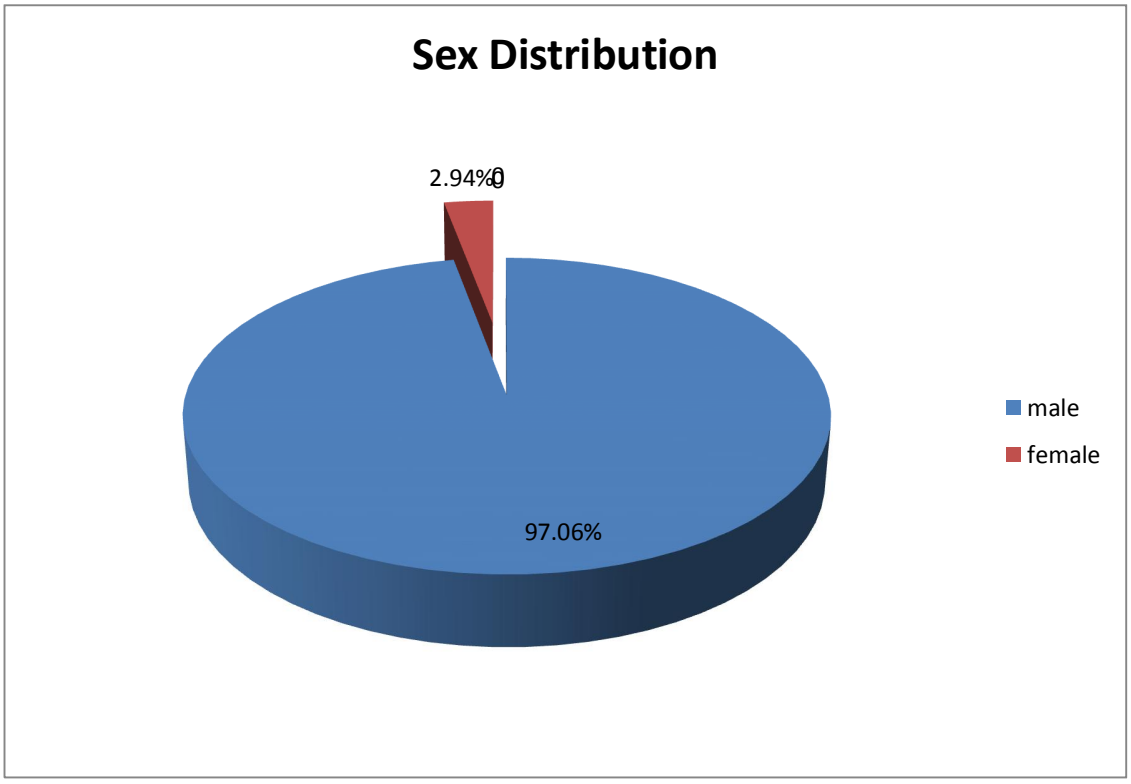


Figure 2: Sex Distribution.

SOCIOECONOMIC STATUS

78 patients (76.47%) belonged to lower socioeconomic status.

PERSONAL HABITS

Of 102 patients 68 patients (66.67%) gave history of smoking, 38 patients were exclusively smokers.

Of 102 patients 34 patients (33.33%) gave history of alcohol intake, 4 patients were exclusively alcoholics.

Of 102 patients 30 patients (29.41%) gave history of both alcohol and smoking.

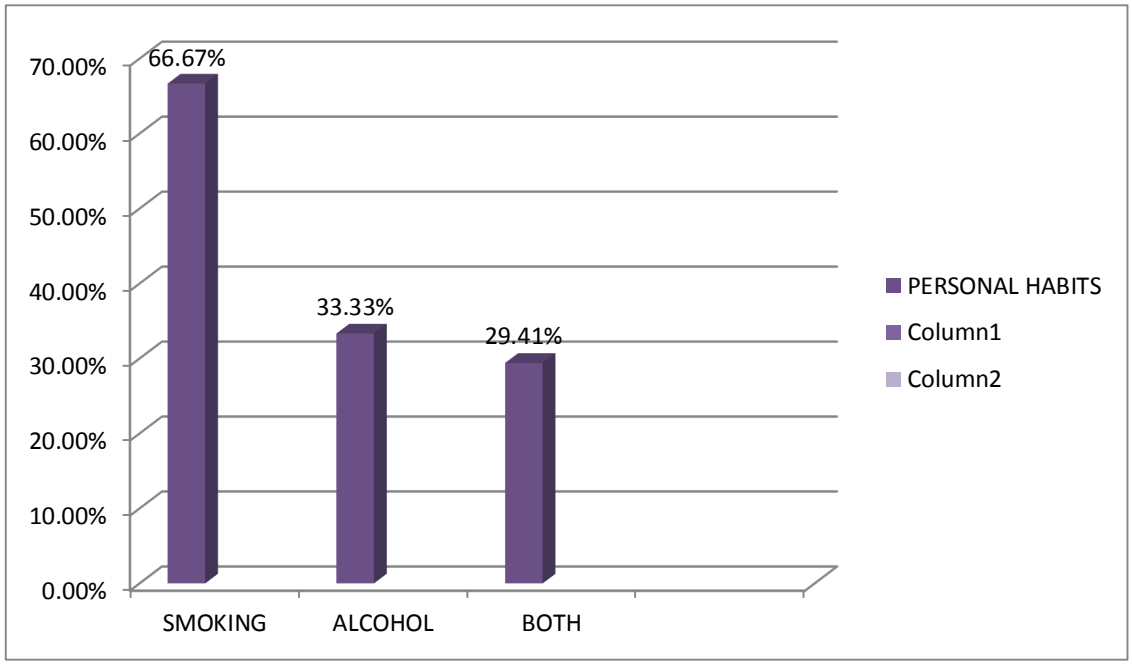


Figure 3: Personal Habits.

HISTORY OF PEPTIC ULCER DISEASE

From our study, 62 patients (60.78%) gave history of peptic ulcer disease

TABLE 3: HISTORY OF PEPTIC ULCER DISEASE

H/O PUD	No. of Case	In %
Present	62	60.78%
Absent	40	39.22%

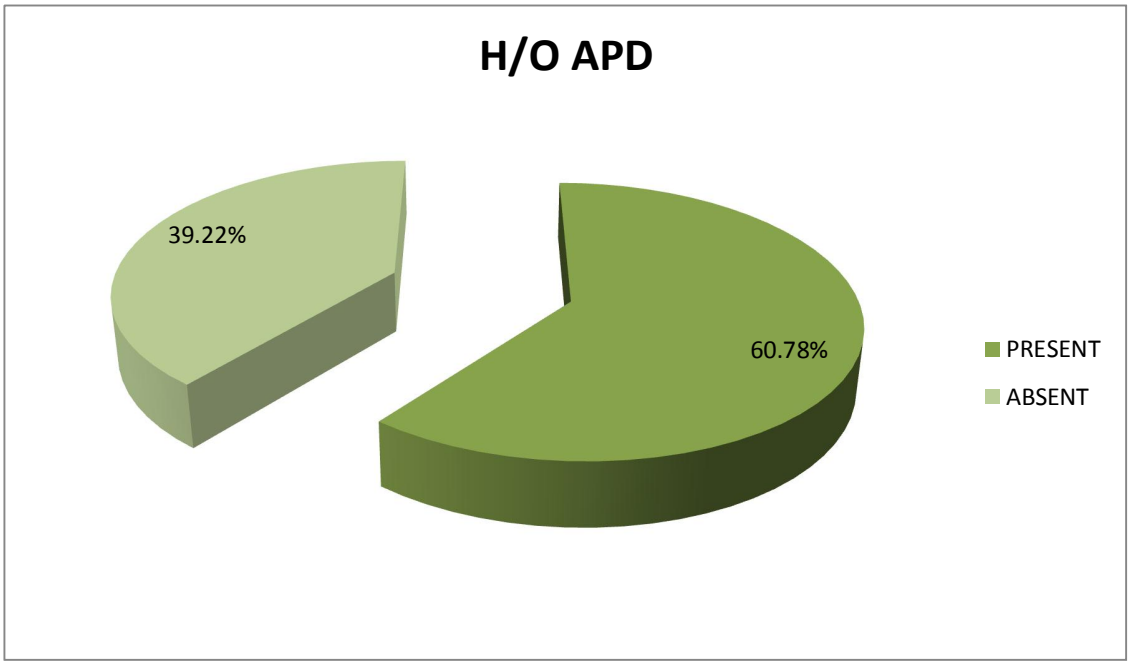


Figure 4: H/O APD.

HISTORY OF DRUG INTAKE (NSAIDS)

26 patients(25.49%) gave history of (NSAIDS) drug intake prior to developing duodenal ulcer perforation (On an average these patients tooks NSAIDS for atleast a period of 1week, one to two weeks prior to the presentation).

Table 4: HISTORY OF DRUG INTAKE (NSAIDS)

Age	No. of Case	Percentage (%)
< 30 Yrs	3	11.54%
30-39 Yrs	1	3.85%
40 - 49 Yrs	4	15.38%
50-59 Yrs	8	30.77%
> 60 Yrs	10	38.46%

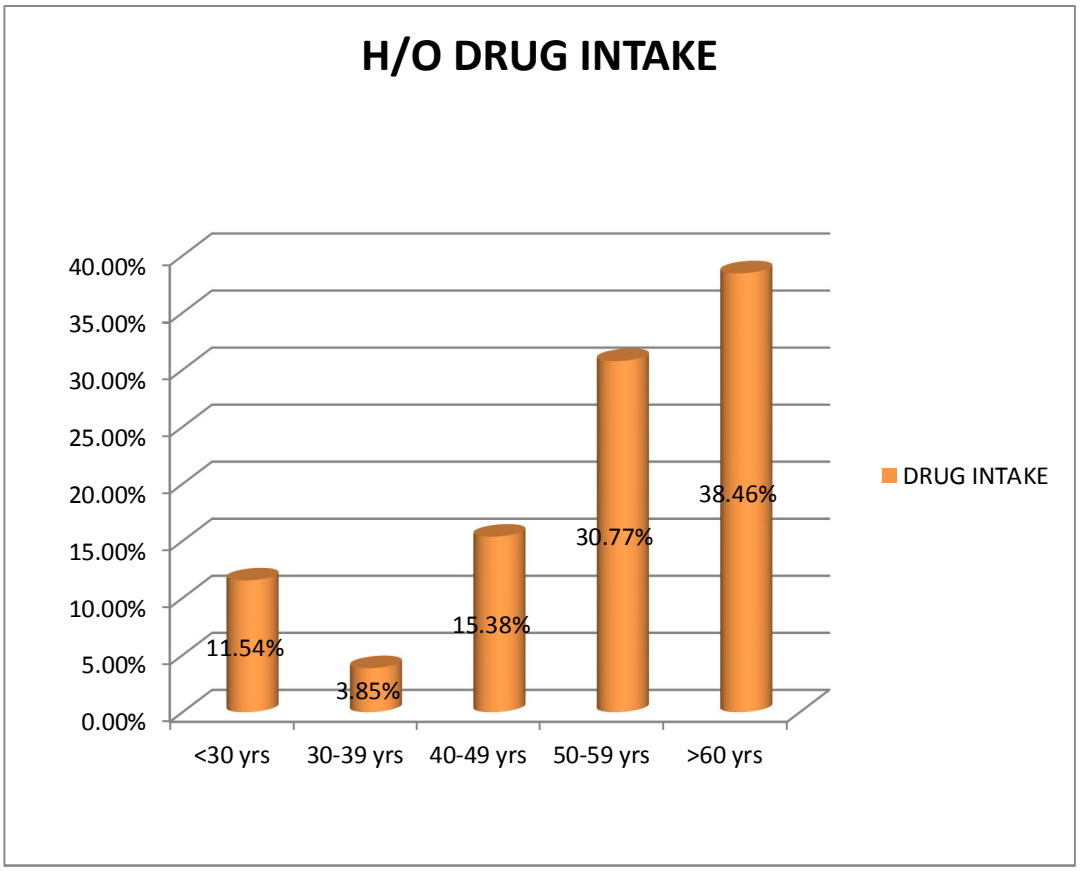


Figure 5: H/O Drug Intake.

SEASONAL TREND

The incidence of cases were more common in winter period.

TABLE 5: SEASONAL TREND

Season	No. of Case	Percentage (%)
Spring	18	17.65%
Summer	23	22.55%
Autumn	23	22.55%
Winter	38	37.25%

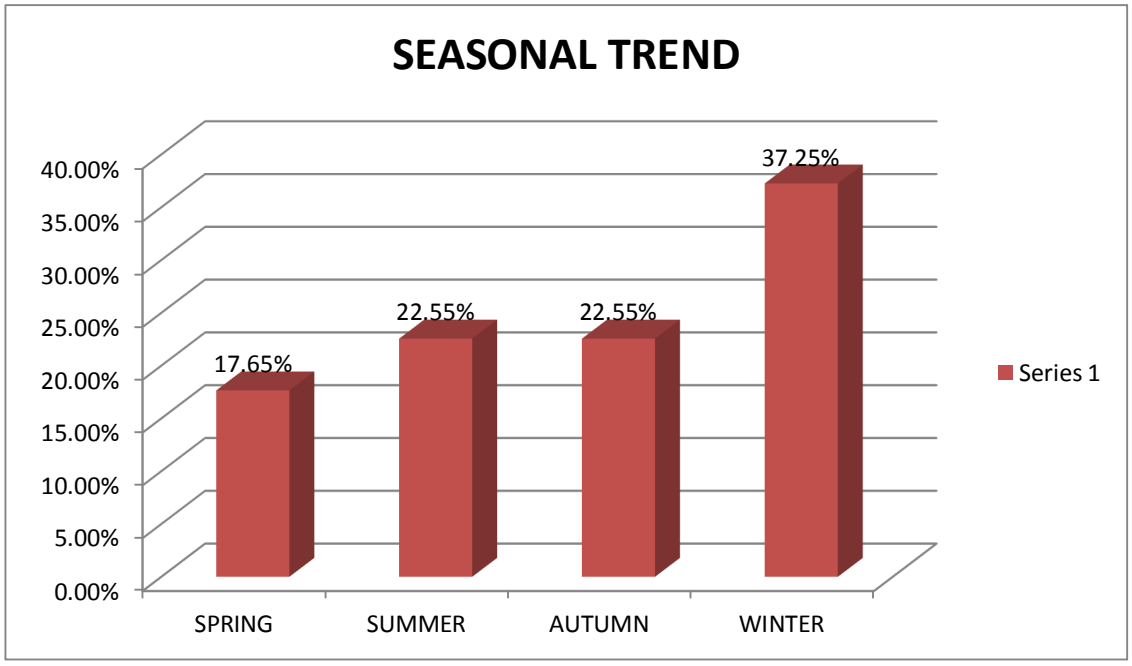


Figure 6: Seasonal Trends

RADIOLOGICAL SIGNS

From our study , 94 patients (92.16%) plain X Ray abdomen showed air under diaphragm .

TABLE 6: X-RAY AIR UNDER DIAPHRAGM

X-ray Air under Diaphragm	No. of Case	In %
Present	94	92.16%
Absent	8	7.84%

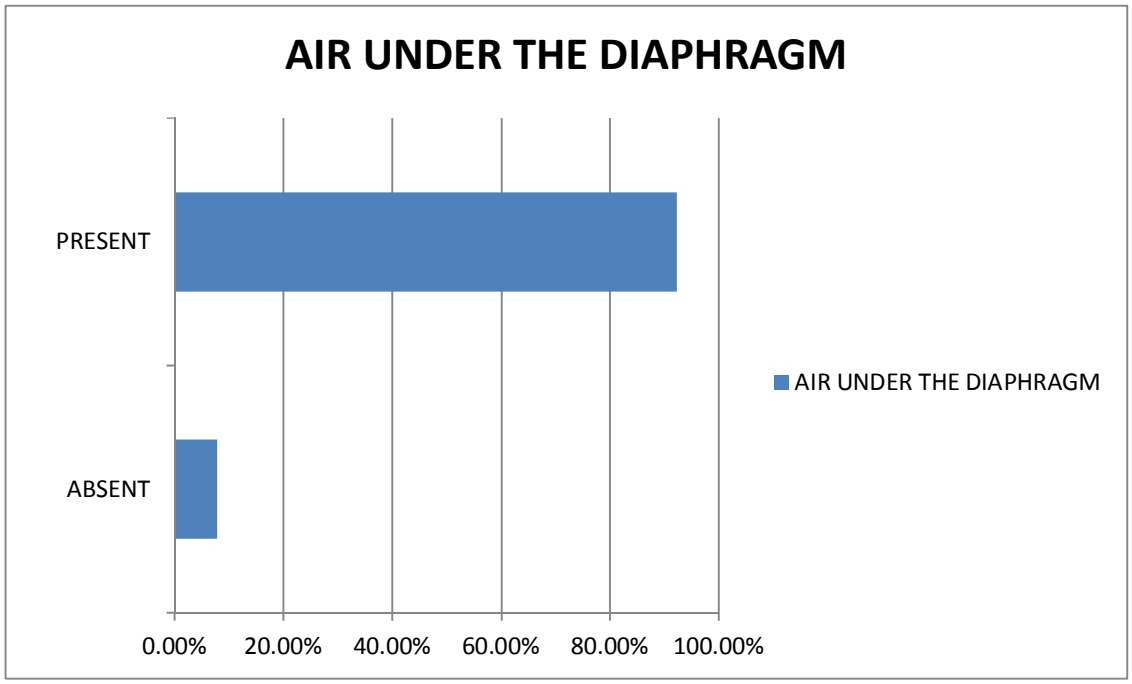


Figure 7: Air Under The Diagphram

MANAGEMENT

- Out of 102 case admitted.
- 99 cases underwent live omental patch closure.
- 1 cases underwent perforation closure with omental patch along with truncal vagotomy with Gastrojejunostomy.
- 1 Case was managed with bilateral flank drain under local anaesthesia because of the patients poor general condition.
- 1 Case was managed conservatively as mentioned above.

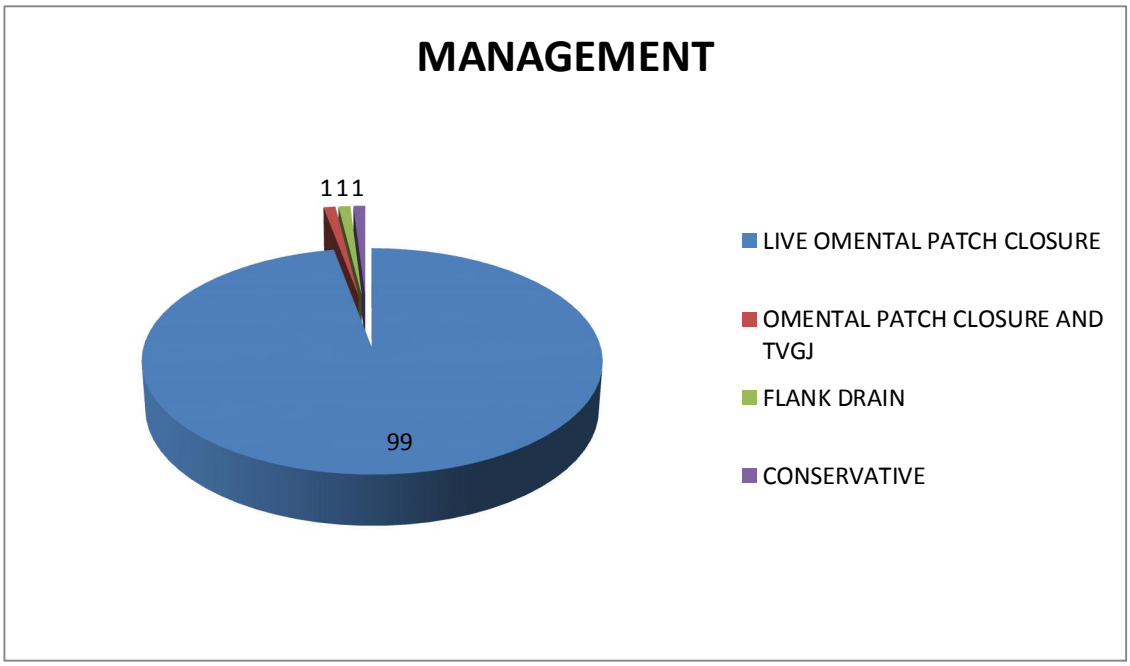


Figure 8: Management.

DURATION OF SYMPTOMS

From our study, the average duration of symptoms for the patients was found to be 1.58 days(1- 3 days).

DELAY

Delay is from the time of admission to Surgical emergency ward upto emergency operation. Average delay is 3.67 hours (2-7 Hrs.)

HOSPITAL STAY

Postoperatively the average duration of stay in hospital was found to be 7.84 days (8-16 days).

POST OPERATIVE COMPLICATIONS

The common complication we encountered was wound infection. Of total 100 cases who underwent emergency surgical management, 13 cases had surgical site infection.

Morbidity Rate from our study was13.00%

MORTALITY

Of total 102 cases who got admitted or managed appropriately , 9 patients expired due to septi-caemia and cardio-respiratory arrest.

Mortality rate from our study was 8.82%.

NON OPERATIVE MANAGEMENT

Three cases who presented with feature with perforation but who had stable vital signs without features of toxaemia were managed conservatively by the following method,

1. Nil per oral - till 5th post op day
2. Naso-gastric Aspiration - till 5th post op day
3. Hourly Pulse Chart, BP Chart - First 48 hrs
4. Abdominal Girth Chart - First 48 hrs
5. Intake / Output Chart - till 5th post op day
6. Intravenous Fluids - till 5th post op day
7. Inj. Pantoprazole 40m g IV bd - till 7th post op day
8. Inj. Cefotaxime 1g IV bd - till 7th post op day
9. Inj. Metronidazole 500mg IV tds - till 7th post op day

   Patients were started on oral diet on 5th post-op day after normal bowel sounds were heard.

   Duration of hospital stay is increased in this group of patients(12days).

DISCUSSION

AGE DISTRIBUTION:

As per age of distribution duodenal ulcer perforation is more common in the age group of > 50 years as per our study.

Taylor et al 25 > 50 years - 50%.

Debakey et al 26 > 50 years - 25%.

From our study > 50 years - 27.45%.

SEX:

Male to female ratio is found to be 33:1.

And on, Desmond Study 6:1.

Rodney Maingot 25:1.

Our study 33:1.

SOCIO-ECONOMIC STATUS:

78 patients (76.47%) belonged to lower socio-economic status.

HISTORY OF DRUG INTAKE:

Patients who are aged (>60 years) and with history of NSAIDs intake are at increased risk of duodenal ulcer perforation. From our study NSAIDs intake was associated with25.49% of patients with Duodenal ulcer perforation of which38.46% of patients were aged > 60 years.

HISTORY OF PEPTIC ULCER DISEASE:

George Stain Study 75% of patients.

Our study 60.78%.

SEASONAL TRENDS:

48 cases (47.06%) of Duodenal ulcer perforation occurred between the period of November to February.

Bloom et al Winter.

Our study Winter.

RADIOLOGICAL SIGNS:

Shaffer Study 70%.

Mann et al 85%.

Our study 94 patients (92.16%).

DURATION OF SYMPTOMS:

The average duration of presenting symptoms was found to be 1.58 days. This was mainly because most of the patients were initially treated at nursing clinics, primary care centres and then referred to our hospital. if they arrived straight to our hospital, they would have been operated earlier and the time lag would have been decreased.

DELAY:

The delay in taking up the patient for emergency operation was 3.67 hrs. Patient who presented with shock needed intense resuscitation and after improving their general condition, they were shifted to emergency operation.

POST-OP COMPLICATIONS:

Patients who presented late (2 days or more) to emergency surgical ward and who had co-morbid illness (Diabetes Mellitus)had increased rate of wound infection.

HOSPITAL STAY:

Delay in presentation to surgical ward contributed to increase in hospital stay by way of wound infection and increased time for improvement in general condition. Increase in age and conservative management also contributed to this.

MORTALITY:

From the study it was found that

1. Age.

2. Associated co-morbid illness.

3. Tim e delay between onset of symptom s and admission to hospital.

4. Patient general condition at the time of presentation, are all important prognostic factors in duodenal ulcer perforation.

of 12 patients who aged more than 60 years, 7 patients had associated co-morbid illness , presented with shock and duration of symptoms > 24hrsMortality rate in this group was 72.72% (Boey et al - 100%).One patient who was managed by bilateral flank drain because of poor general condition, died. The Study on outcome of the non-operative management was not carried out because patients were not randomized.

CONCLUSION

The conclusion from our study is

1. Duodenal ulcer perforation is becoming more common in the age group > 50 years.

2. Associated risk factors are found to be smoking, alcohol intake and previous history of acid peptic disease.

3. NSAIDs intake are associated with increased risk of perforation commonly in older age group (> 60 years).

4. Wound infection is the common post-Op complication encountered.

5. Mortality rate is 8.82% in our study.

6. Prognostic factors are

    1) age.

    2) Co-morbid illness.

    3) Duration of symptoms.

    4) Patient general condition at the time of presentation.

SUMMARY

- One hundred and two cases of Duodenal ulcer perforation were studied.In all 100 cases underwent Laparotomy and the perforation was found in the anterior aspect of first part of the duodenum.

- From our study Male to Female ratio was 33:1. With 78 patients (76.47%) belonged to lower socioeconomic status. Of 102 patients 68 patients (66.67%) gave history of smoking, 38 patients were exclusively smokers. And 62 patients (60.78%) gave history of peptic ulcer disease.

- Then 26 patients(25.49%) gave history of (NSAIDS) drug intake prior to developing duodenal ulcer perforation. The incidence of cases were more common in winter period.

- Duodenal ulcer perforation is becoming more common in the age group > 50 years. Mortality rate is 8.82% in our study.

BIBLIOGRAPHY

1. EL - Serag HB, Sonnenberg A, : Opposing time trends of peptic ulcer and reflux disease Gut 43 :327, 1998.

2. Maingot's Abdominal operations - 10th ed. Haile T.Debas, Sean J. M olvitill, Appleton & Lange Vol. I :pp 983 - 984.

3. Gunshefski L, Flancbaum L, Brolin RE,Frankel A,Changing patterns in perforated peptic ulcer disease.Am Surg. 1990 : 56 (4) : 270 - 4.

4. Flemstorm G, Garner A, Gastro-duodenal HCO3 transport ; characteristics and proposed role in acidity regulation and mucosal protection. AmJPhysiol 242 ; G 183 - G 193, 1982.

5. Hogan DL, Rapier RC,Dreligner A, et. al., Duodenal bicarbonate secretion; Eradication of H. Pylori and duodenal structure and functions in humans. Gastro-enterology 110: 705 - 716 , 1996.

6. Chen MC, Chang A, Bobl T et. al. Apical Acidification induces para-cellular injury in canine gastric mucosal layer AmJ. Physiol 267; G 1012-G 1020, 1994.

7. Schoen RT, Vender RJ. Mechanisms of NSAID -induced gastric damage - AmJ Med 86; 449 - 458,1989.

8. Graham Dy, Lidsky M D, Cox Am , et al; Long Term NSAID use and H. Pylori infection Gastro-enterology100 : 1653 - 1657, 1991.

9. Svanes C, Soride JA, Skarstein A, et. al., smoking and ulcer perforation Gut. 2 : 177 - 180, 1997

10. Bonneive O Causes of death in duodenal and gastric ulcer Gastro-enterology 73 : 1000 - 1004,1977.

11. Friedman G D, Siegelaub AB, Seltzer CC, Cigarettes,alcohol, coffee and peptic ulcer. N Eng J. M ed 290 -469 - 473, 1974.

12. Mc Arthur K, Hogean D, Isenberg JI, Relative stimulatory effects of commonly ingested beverages on gastric acid secretion in humans. Gastro-enterology 83;199 - 203, 1982.

13. Longamn MJ, Cooke AR : Gastric and duodenal ulcer and their associated disease, Lancet 1;680 - 683 - 1976,

14. Kirk AP, Dooley JS, Hunt RH : Peptic ulceration in patients with chronic liver disease. Dig Dis Sci. 25;756 - 760, 1980.

15. Feldman M, Mental stress and peptic ulcers : An earth shaking association. AmJ Gastro 93 ; 291-292,1998.

16. Asaka M , Kimura T, Kudo M , et al : Relationship of H. Pylori to serum pepsinogens in anasymptomatic Japanese population. Gastro-enterology 102 ; 760 -766, 1992.

17. Boren T, Falk P, Roth KA, et al., Attachment of H.Pylori to human gastric epithelium mediated by blood group antigens, science 262 : 1892 - 1895, 1993.

18. Sleisenger & Fordtrans, Gastro-intestinal and liver disease Mark Feldman, Lawrence, J. Brandt (eds);Saunders Vol I. pp 1099 - 1102.

19. Crofts TJ, park KG, Steele RJ, et al., A randomized trail of non-operative treatment for perforated peptic ulcer N Eng J med 1989 ; 320 (15) ; 970 - 3.

20. Svanes C, Trends in perforated peptic ulcer, Incidence, etiology treatment and prognosis. World J.Sug. 24 : 277 - 283, 2000.

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H/O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
1	MARUDHU	55	M	+/-	1	HT	+	+	-	103	+	Live OPC	-	9
2	RAJA	42	M	+/-	1	-	-	+	-	98	+	Live OPC	-	8
3	KUMAR	28	M	+/-	1	-	-	-	-	87	+	Live OPC	-	8
4	ARUN	42	M	+/-	3	DM	-	-	+	110	+	Live OPC	WI/Death 4th POD	-
5	MANNAR	38	M	+/-	2	-	-	+	-	99	+	Live OPC	-	8
6	AYANAR	44	M	+/-	2	-	-	-	-	90	+	Live OPC	-	8
7	SHANMUGAM	46	M	+/-	1	-	-	+	-	89	+	Live OPC	-	8
8	KUTTY	44	M	+/-	1	-	-	+	-	85	+	Live OPC	-	8
9	CHINAIYA	37	M	+/-	1	-	-	-	-	98	+	Live OPC	-	8
10	MUTHU	23	M	+/-	3	-	+	+	+	111	+	Live OPC	Death 1st POD	-
11	MURUGAN	36	M	+/-	2	-	-	-	-	107	-	Live OPC	-	9
12	PALANI	47	M	+/-	1	-	-	+	-	98	+	Live OPC	-	10
13	NATAESAN	56	M	+/-	2	DM	-	+	-	97	+	Live OPC	WI	14
14	RAMU	48	M	+/-	1	-	-	-	-	102	+	Live OPC	-	8
15	JAGAN	28	M	+/-	1	-	+	+	-	120	+	Live OPC	-	8

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H/O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
16	AYYAN	58	M	+/-	1	HT	+	+	-	89	+	Live OPC	WI	8
17	EESAN	69	M	+/-	3	IHD+DM	-	-	+	118	+	Live OPC	Death 2nd POD	-
18	RAJASEKAR	33	M	+/-	1	-	-	+	-	90	+	Live OPC	-	8
19	THIRUMALAI	48	M	+/-	1	-	-	+	-	89	+	Live OPC	-	8
20	URUMY	56	M	+/-	2	-	-	-	-	95	+	Live OPC	-	8
21	ILAIYARAJA	42	M	+/-	2	-	+	+	-	97	+	Live OPC	-	9
22	PONNAN	27	M	+/-	1	-	-	+	-	93	-	Live OPC	-	9
23	ARJUNAN	34	M	+/-	1	DM		+	-	92	+	Live OPC	WI	12
24	KARPAGAM	63	F	+/-	2	DM	+	-	+	116	+	Live OPC	Death 1st POD	-
25	DURAI	45	M	+/-	3	-	-	+	-	98	+	Live OPC	-	8
26	FAZIL	35	M	+/-	3	-	-	-	-	96	+	Live OPC	-	9
27	GODHANDAM	46	M	+/-	1	-	-	+	-	87	+	Live OPC	-	8
28	HARI	28	M	+/-	1	-	-	-	-	84	-	Live OPC	-	8
29	JEYARAJ	48	M	+/-	1	-	-	-	-	89	+	Live OPC	-	8
30	KRISHNAN	67	M	+/-	1	DM	+	+	-	98	+	Live OPC	-	8

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H /O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
31	LAKSHMANAN	65	M	+/-	3	-	+	+	+	98	+	Live OPC	Death 2nd POD	-
32	CHANDRU	46	M	+/-	1	-	-	+	-	87	+	Live OPC	-	8
33	VIJAYAN	22	M	+/-	2	-	-	-	-	89	+	Live OPC	-	8
34	BABU	32	M	+/-	2	-	-	-	-	106	+	Live OPC	-	8
35	NAGARAJ	58	M	+/-	1	-	+	+	-	95	-	Live OPC	-	9
36	MURUGAESAN	44	M	+/-	1	DM	+	+	-	100	+	Live OPC	WI	14
37	MURALI	39	M	+/-	3	-	-	-	+	101	+	Live OPC	Death 3rd POD	-
38	ELAMARAN	66	M	+/-	1	HT+DM	+	+	-	89	+	Live OPC	WI	13
39	RAMESH	26	M	+/-	1	-	-	-	-	98	+	Live OPC	-	9
40	THOTTI	38	M	+/-	1	-	-	+	-	98	+	Live OPC	-	9
41	YEGNARAJ	56	M	+/-	1	-	-	-	-	87	+	Live OPC	-	8
42	UMESH	48	M	+/-	1	-	-	-	-	98	+	Live OPC	-	8
43	ILAMARAN	68	M	+/-	2	HT	+	+	+	90	+	Live OPC	-	8
44	PARAMU	54	M	+/-	2	HT	+	+	-	109	+	Live OPC	-	8
45	ARASAN	28	M	+/-	3	-	-	+	+	120	+	Live OPC	-	8

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H/O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
46	SURIYA	23	M	+/-	2	-	-	+	-	99	+	Live OPC	-	8
47	DEVA	34	M	+/-	2	-	-	-	-	96	-	TVGJ	-	9
48	GURU	55	M	+/-	1	HT+DM	+	+	-	90	+	Live OPC	WI	10
49	HARISH	46	M	+/-	1	-	-	-	-	94	+	Live OPC	-	8
50	JEYAPAUL	36	M	+/-	1	-	+	+	-	93	+	Live OPC	-	9
51	KESAVAN	25	M	+/-	2	-	-	-	-	92	+	Live OPC	-	9
52	LAKSHAN	65	M	+/-	3	DM	+	+	+	112	+	Live OPC	WI	16
53	MALLIGA	44	F	+/-	1	-	+	+	-	105	+	Live OPC	-	8
54	VIMAL	53	M	+/-	1	DM	-	+	-	102	+	Live OPC	-	8
55	BARANI	37	M	+/-	1	-	-	+	-	98	+	Live OPC	-	8
56	NARAYANAN	45	M	+/-	2	-	-	-	-	89	+	Live OPC	-	8
57	MANI	43	M	+/-	2	-	-	+	-	86	+	Live OPC	-	8
58	ARUN	46	M	+/-	3	-	-	+	+	106	+	Live OPC	-	9
59	BALA	44	M	+/-	1	-	-	-	-	98	+	Live OPC	-	7
60	GOPAL	66	M	+/-	2	DM	+	+	+	130	+	Live OPC	WI	11

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H/O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
61	ANBU	24	M	+/-	1	-	-	+	-	109	+	Live OPC	-	8
62	ASHOK	47	M	+/-	1	-	-	-	-	98	+	Live OPC	-	8
63	VIGNESH	29	M	+/-	1	-	+	+	-	89	+	Live OPC	-	10
64	SRINATH	37	M	+/-	2	-	-	+	-	97	+	Live OPC	-	9
65	SENTHIL	48	M	+/-	2	-		-	-	97	+	Live OPC	-	8
66	RAVI	62	M	+/-	3	HT+DM	+	+	+	108	+	B/L flank drain	Death 2nd POD	-
67	KIRUBA	28	M	+/-	1	-	-	-	-	98	+	Live OPC	-	9
68	KARTHICK	54	M	+/-	1	-	+	+	-	98	-	Live OPC	-	8
69	MANIVANAN	48	M	+/-	2	-	-	+	-	98	+	Live OPC	-	8
70	RAGUPATHY	38	M	+/-	1	-	-	+	-	89	+	Live OPC	-	8
71	ARASU	54	M	+/-	1	IHD+HT	-	+	-	94	+	Live OPC	WI	14
72	THANGAMANI	58	M	+/-	2	-	-	-	+	112	+	Live OPC	-	9
73	PERIYASAMY	61	M	+/-	1	-	-	-	-	89	+	Live OPC	-	8
74	MUTRHURAJ	36	M	+/-	1	-	-	+	-	98	+	Live OPC	-	8
75	SRINIVASAN	22	M	+/-	1	-	-	+	-	95	+	Live OPC	-	8

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H/O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
76	PACHAIPONDY	48	M	+/-	2	-	-	+	-	95	+	Live OPC	-	9
77	GOKUL	39	M	+/-	2	-	-	+	-	95	+	Live OPC	-	7
78	PANDY	28	M	+/-	1	-	-	-	-	96	+	Live OPC	-	9
79	CHANDRAN	56	M	+/-	1	HT+DM	+	+	-	93	+	Live OPC	WI	16
80	SUBRAMANI	29	M	+/-	2	-	-	-	-	90	+	Live OPC	-	8
81	AZHAGIRI	64	M	+/-	3	-	+	+	+	106	+	Live OPC	Death 1st POD	-
82	SANKARAN	37	M	+/-	1	-	-	-	-	86	+	Live OPC	-	8
83	EZHILRAJ	26	M	+/-	1	-	-	+	-	90	+	Live OPC	-	8
84	JAYABALAN	19	M	+/-	1	-	-	-	-	86	+	Live OPC	-	8
85	AROKIARAJ	24	M	+/-	1	-	-	+	-	90	+	Live OPC	-	9
86	BASKARAN	48	M	+/-	2	-	-	+	-	98	+	Live OPC	-	8
87	KANNAN	58	M	+/-	3	-	-	+	-	85	-	Live OPC	-	8
88	NATARAJ	35	M	+/-	1	-	-	-	-	86	+	Live OPC	-	8
89	ANANDH	45	M	+/-	2	-	+	+	-	97	+	Live OPC	-	9
90	SEKAR	56	M	+/-	2	HT	-	+	-	94	+	Live OPC	WI	14

MASTER CHART

SL.NO	NAME	AGE	SEX	PAIN/ DISTENSION	DURATION	CO-MORBID ILLNESS	H/O DRUG INTAKE	H/O APD	SHOCK	PULSE RATE	AIR UNDER THE DIAPHRAGM	PROCEDURE	POST-OP COMPLICATIONS	HOSPITAL STAY IN DAYS
91	GEORGE	44	M	+/-	1	DM	-	-	-	96	+	Live OPC	WI	13
92	MOHAN	28	M	+/-	2	-	-	+	-	95	+	Live OPC	-	8
93	SELVAM	46	M	+/-	2	-	-	-	-	94	+	Live OPC	-	8
94	SINGARAVELU	22	M	+/-	1	-	-	+	-	94	+	Live OPC	-	9
95	ARAVINDH	25	M	+/-	1	-	-	-	-	97	-	conservative	-	10
96	NALLAMUTHU	43	M	+/-	1	-	-	-	-	87	+	Live OPC	-	9
97	NAGALINGAM	47	M	+/-	2	DM	-	-	-	83	+	Live OPC	-	8
98	ETHIRAJ	37	M	+/-	1	-	-	+	-	98	+	Live OPC	-	8
99	SAKTHI	42	M	+/-	2	-	-	-	-	109	+	Live OPC	-	8
100	MARIYAMMA	54	F	+/-	1	IHD+DM	+	-	-	108	+	Live OPC	WI	16
101	SELVARAJ	26	M	+/-	2	-	-	+	-	78	+	Live OPC	-	9
102	KOTTY	68	M	+/-	3	DM	+	+	+	90	+	Live OPC	Death 1st POD	-